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Original Communications

EVARTS AMBROSE GRAHAM (1883-1957)

Eulogy

Tom Burford

ON MARCH 4, 1957, the spirit of Evarts Ambrose Graham passed quietly into that select Valhalla that shelters the shades of the truly great. Death with tragic irony resulted from bronchogenic carcinoma, a disease he had studied for over a quarter of a century, and a malady which he had battled so valiantly and successfully to conquer and control in others. Insidious in onset and hopeless at the time of discovery, his disease progressed with merciful rapidity. Aware from the beginning of the diagnosis and its terrible portent, he faced the issue with awe-inspiring equanimity. Not once did he manifest the slightest degree of bitterness or self-pity. With serene objectivity he sublimated all concern for self to an amazingly critical contemplation of cancer in general, and of lung cancer in Evarts Graham in particular. He was much more concerned by the fact that he, a nonsmoker for seven years, in developing the disease might have invalidated his own etiological contentions than he was by the prospect of dying.

Born in Chicago on March 19, 1883, he attended public schools and the Lewis Institute. He graduated from Princeton in 1904 where early in his undergraduate period he decided to devote his life to a surgical career. It was at Princeton that he stated his oft-quoted determination "To do major surgery, to engage in research work, and to have a clinic of younger men who would be interested in studying and developing ideas." This cogent capsule contains the essence of the great surgical saga that was the life and career

Portions of this Eulogy were presented before the Society of Clinical Surgery in New York in October, 1957, but not published.

of Evarts Graham. All of the fine panegyrics that will be penned at the end can add little to what he so confidently said of himself at the beginning. In 1907 he received his M.D. from Rush Medical College where his father, Dr. David W. Graham, was a teacher of surgery. Following graduation he interned at the Presbyterian Hospital in Chicago for one year. He spent the following year as a Fellow in Pathology at Rush. Dissatisfied with the traditional surgical training of that era, he did special work in chemistry and physiology. Working with Woodyatt, Carlson, Wells, and others, he acquired a broad background in the basic disciplines which did much to inspire and shape his later career. It was during this period that he met Helen Tredway, a graduate student in chemistry. They were married in 1916. Dame fortune smiled often on Evarts Graham, but never more warmly than when this gracious and brilliant lady agreed to share his life. She created the cultured orderly home so necessary to the success of a busy scientist and surgeon on the one hand, and on the other carved out for herself a distinguished career in teaching and research.

In 1915 he entered private practice. He rarely spoke of this experience and it is noteworthy that he was careful to omit it from his Curriculum Vitae. However, with his infallible gift of serendipity, even this experience was to prove significant. First-hand contact with unnecesary surgery, ghost surgery and fee-splitting shocked and infuriated him. Not long before his death in discussing his early years with me he said of this awakening, "I resolved then that if I ever had a chance to do something about these things that I would." Those who were privileged to serve with him on the Board of Regents of the American College of Surgeons know how effectively he kept that resolution.

In 1918 he entered the United States Army with the rank of Captain. Later in the same year he was appointed to the Empyema Commission and promoted to the rank of Major. It was while a member of the Empyema Commission that he did his brilliant work on the dynamics of intrapleural pressures which led to a safe and rational approach to the problem of intrapleural sepsis. He was then sent to France as commanding officer of the 34th Evacuation Hospital. On his return to the States he was sought out by Philip Shaffer, Canby Robinson, and Eugene Opie and offered the Bixby Chair of Surgery at the Washington University School of Medicine in St. Louis, which he accepted. It is a matter of record that he took over his duties as professor of surgery wearing his army uniform. It cannot be verified, however, as one disgruntled, and perhaps bruised, contemporary contended, that he also wore spurs. In St. Louis he joined with Shaffer, Erlanger, Robinson, Marriott, Opie, and others in the exciting adventure of creating a great medical school. In a short time he was the acknowledged leader of this distinguished group, and the school's international reputation today is due more to the efforts of Evarts Graham than to any other member of the faculty. During his tenure the surgical service was a world-renowned Mecca.

In 1922 he first conceived the idea that visualization of the gall bladder might be possible. His broad background in chemistry made him aware that

the chlorinated phenolphthaleins are excreted almost exclusively in bile. By substituting radiopaque atoms to these compounds he hypothesized that visualization of the biliary tract should be possible. The epochal success of his ensuing experiments and the success of cholecystography are known to all. This, in my opinion, was his greatest scientific contribution, and was, as Alfred Blalock has stated, worth a Nobel Prize. On April 5, 1933, he made one of the great technical contributions of all times when he performed the first successful one-stage pneumonectomy for bronchogenic carcinoma. To some it has seemed strange that Graham, with his emphasis on basic disciplines, should consider a purely technical feat his greatest contribution. To understand this apparent paradox it is necesary to have known the man. He came to St. Louis noted only for productive research and was contemptuously dubbed the "Mouse Surgeon" by the entrenched, but now forgotten, old guard. He neither forgot nor forgave this indignity. In addition, he was always mindful that careful, bloodless techniques were not his forte. How natural then that the fait accompli of so magnificent a technical feat should symbolize to him a major victory—a triumph in the only area of his many endeavors where he ever felt the slightest sense of inadequacy.

Each year brought increasing prestige and heavier burdens of work and responsibility. His capacity for sustained painstaking effort was prodigious. This attribute plus his uncompromising integrity and his supreme self-confidence were the keystones, in my opinion, of his character and career.

Throughout his notable career he was untiring in his efforts to improve the standards of medical care and medical education and to this end he unhesitatingly accepted appointments to many national committees. He served for fourteen years as a member of the Medical Fellowship Board of the National Research Council. From 1940 to 1946 he was Chairman of the Committee on Surgery of the National Research Council. In 1944 and 1945 he was a member of the Commission on Hospital Care. In 1952 he was appointed a member of the President's Commission on the Health Needs of the Nation. In 1953 and 1954 he served on the Medical Task Force of the first Hoover Commission. In each instance he served with the courage and effectiveness that characterized every phase of his career.

Honors and distinctions of every kind were showered on Evarts Graham. Recipient of fourteen honorary degrees and many distinctions, he was repeatedly honored at home and abroad. No American surgeon has ever so completely won the esteem and affection of Colleagues in South America, the Continent, and in England in particular. Even after retirement the pilgrimage of notables from the four corners of the world continued to his office—a tribute to the acknowledged Dean of World Surgery.

He was elected directly to active membership in this Association at the third annual meeting in New Orleans in 1920, becoming the eighty-third member. In 1928 he was elected President. In 1931 with the establishment of the JOURNAL OF THORACIC SURGERY he became the first editor of the JOURNAL, a post he held with conspicuous success until his death. In 1952 he became a

senior member and, in 1955, on the motion of Leo Eloesser, became the twenty-fifth honorary member of the Association. While not one of the founding fathers, his contributions to this body and his influence in shaping the destiny of this organization rank second to none. By honoring Evarts Graham, the American Association for Thoracic Surgery acknowledges its indebtedness to its greatest member and thereby does honor unto itself.

Retirement came to Graham in 1951. Whatever academic retirement may mean to lesser men, it was to him a challenge and a new beginning. He returned to the laboratory with the zest of a young graduate student, and began a large-scale investigative program. His studies ranged from the experimental production of cancer in mice with the tars from cigarette smoke to an effort to determine why the antlers of the deer family are regenerated so rapidly. Outside the laboratory his interests seemed to widen rather than contract.

From 1951 to 1954 he was Chairman of the Board of Regents of the American College of Surgeons, and in 1955 was elected President of the Sixteenth Congress of the International Society of Surgery.

He was among the first to become intelligently alarmed by the problem of radioactive fall-out. His reaction was characteristic. He moved directly into the national political arena, and it was due in no small measure to the efforts of Graham and his friend Norman Cousins that Adlai Stevenson was persuaded to make a campaign issue of Strontium 90, and nuclear testing.

Socially and politically, Graham was an enlightened and often militant liberal—a born crusader. Emotionally and ethically he was an unreformed Victorian. To him privilege and power implied stern responsibility. "He knew no motive but interest—no criterion but success." It was thus inevitable that his rise to high position should foster within him a sense of noblesse oblige, and that this should make men of little discernment often falsely call him cold and autocratic and even ruthless. His biographer will have no difficulty in showing up the superficiality of these evaluations and will find an abundance of warm color, generous tones, and even humble shades from which to paint a true picture of Graham the man.

Gordon Taylor could well have been speaking of Graham when he said of Moynihan in the first Leeds Lecture, "Those who have been reckoned great in surgery must of necessity be supremely skilled in the craftsmanship of our profession; they must, by reason of the initiation of some novel and successful system of treatment, have conferred untold blessings on mankind; their work must have illuminated the dark, mysterious chasms in our knowledge of disease. It is necessary that they should have inspired their pupils and their surgical brethren with their own ideals, and should have inaugurated or sponsored some policy which has advanced the science of surgery. Their prospects of pre-eminence . . . are perhaps enhanced, if they are accounted 'Men of Affairs,' and if they command success in their everyday relationships and dealings with those who are not necessarily linked by close ties with our profession."

By these and by all criteria Evarts Graham will be reckoned among the greatest.

PRESIDENTIAL ADDRESS
A CASE REPORT AND MISCELLANEOUS COMMENTS

Brian Blades, M.D., Washington, D. C.

MY FIRST and pleasant obligation is to express profound appreciation to the members of The American Association for Thoracic Surgery. And of equal importance, to thank the Officers and committee members whose efforts determine the success or failure of our meeting. Particular gratitude is deserved by the Secretary, Dr. Hiram Langston, and Miss Ada Hanvey, Administrative Assistant.

The honor of being President of this Association is impossible to define. The endeavors of those whom I have just cited have made most of the duties and responsibilities of the office easy. There is, however, one notable exception, the preparation and presentation of the presidential address.

Mild plagiarism was first considered. A careful review of my predecessors' contributions, however, made this impractical. Their subjects have consisted of dissertations on philosophical, educational, and historical matters or reports of brilliant surgical achievements or research endeavors. It became apparent I had neither information nor the ability to imitate their examples.

In desperation I have decided to present a case report as the principal feature of this address.

The unusual history, methods of diagnosis and, finally, the surgical management will, I hope, justify the review of a clinical problem in a Presidential Address.

Following the case report there will be a few miscellaneous and probably confusing comments.

From the Department of Surgery, The George Washington University, Washington, D. C.

Presented at the Thirty-eighth Annual Meeting of The American Association for Thoracic Surgery at Boston, Mass., May 16-18, 1958.

CASE REPORT*

BARNES HOSPITAL

History No. 37532

Name Gilmore, James Lee (Dr.) Nee

Address 1331 Inverness Pittsburg Pennsylvania

Age (1st Admission) 49 Years Married, Single, Divorced Separated

Sex Male Race White Nativity Pennsylvania Occupation Physician

Nearest Relative or Friend Marjorie Gilmore How Related Wife

Address Same

Family Physician

Address

Recommending Physician Dr. W. T. Mitchell, Jr. O.P.D. No.

Address Pittsburg Pennsylvania

Staff Physician Dr. Graham Dr. Arbuckle Dr. Singer

Remarks

*The case report is a facsimile of the original records including misspelled words.

Adm. Date	Serv.	Disch. Date	Diagnosis & Operation	Condition At Discharge
27 Feb 33	Surg Med	5 Mar 33	Tumor of Bronchus, Uncertified. (Lt. upper lobe) Artificial Pneumothorax. (Carcinoma?) 3-1-33 Bronchoscopy.	Not Improved
9 Mar 33	Surg	9 Mar 33	Tumor of Bronchus, Uncertified. (Carcinoma?)	Not Treated
13 Mar 33	Med Surg	24 Mar 33	Carcinoma of Bronchus. 3-14-33 Bronchoscopy. Excision of Tissue of Bronchus for Diagnosis. 3-21-33 Bronchoscopy.	Not Treated
4 Apr 33	Surg	18 Jun 33	Carcinoma of Bronchus. (Lt. upper lobe) Carcinoma of Lung. (Squamous cell) 4-5-33 Pneumectomy. (Total) (Left) Blood Transfusion. Thoracoplasty. (Partial) (Left) 4-19-33 Thoracostomy. (Closed drainage) (Left) 5-13-33 Thoracostomy. (for empyema) 5-22-33 Thoracoplasty. (Partial) (Left)	Improved

Abstract of the record of Dr. James L. Gilmore. Age 48 yrs.

Admitted	Discharged
2-27-33	3-5-33
3-9-33	3-9-33
3-13-33	3-24-33
4-4-33	6-18-33

Patient enters hospital with a history of:

1. Repeated attacks of cough and fever with pain in the chest.
2. General lassitude.
3. Easily fatigued.
4. Loss of weight and inability to gain weight.

} 7 months duration.

In January 1929 patient developed pneumonia of the right lower lobe. It spread from this location and involved the entire lung. Convalescence was delayed for several weeks and terminated by his "raising some pus."

In July 1932 he experienced a general malaise with chilly sensation and a fever of 104. Nothing found on P. X. WBC. 17,000. On Aug. 11th an x-ray revealed a fan-shaped shadow with the base outward, in the region of the left axilla. By Aug. 20th his symptoms had subsided and the x-ray shadow had become smaller. On Oct. 7th he experienced a repetition of his former symptoms with return of the former x-ray shadow. This subsided in a few days only to recur again about Oct. 20th. At this time there was some dullness and a diagnosis of (interlobar empyema) lung abscess was made. On attempting to aspirate it (Dec. 5, 1932) a pneumothorax developed, subsequent to which his symptoms improved. He experienced some pain in the left chest following production of pneumothorax (not much pain and no bloody sputum at any time). The pneumothorax was continued and was improved until 10 days before admission (2-17-33) when he had a recurrence of fever and discomfort, etc. Examination revealed a man of medium build with suggestion of loss of weight and having a pale pasty complexion. Did not look acutely ill. The left chest moved less than the right and B. S. were diminished or absent. X-rays of the chest showed the left upper lobe atelectatic with pneumothorax present. (The lower lobe seemed fully expanded and adherent to the chest wall). RBC. 4,800,000, WBC. 11,500, HB. 85%.

In view of the patient's history, PX., and x-ray findings, a tentative diagnosis of bronchial obstruction by a tumor was made.

Bronchography substantiated the diagnosis of bronchial obstruction of the left upper lobe.

Bronchoscopy 3-1-33 revealed tissue not unlike the appearance of granulation tissue, closing off the lt. upper bronchus. Bronchoscopy repeated on 3-14-33 and 3-21-33. Biopsy taken revealed squamous cell carcinoma of the bronchus.

Patient took microscopic slides to home pathologist to have verified.

Patient was readmitted 4-4-33 for lobectomy. At the operation (4-5-33) the tumor and suppurative process was found such that it was necessary to remove the entire left lung. Many adhesions made this somewhat difficult. Seven ribs were also removed, 3 to 9 inclusively. Left the operating room in good condition: (had received glucose-acacia and some blood). Closed drainage yielded several (800 to 1000 cc.) of serosanguinous exudate for 48 hours. There was little P.O. reaction except for some dyspnea especially on exertion. Considerable deep-seated pain was experienced at times but was fairly well controlled. Some infection of the unobliterated portion of the left chest occurred on the 8th or 9th P.O. day which was drained by a stab wound posteriorly. This failed to function after 2 weeks (during which time patient was being prepared for removal of the remaining 1st and 2nd ribs to obliterate the remaining space) so a 2nd drain was placed

anteriorly through the 1st interspace. The 1st and 2nd ribs were removed on 5-22-33, with little post-operative reaction. His pain then completely subsided and within 3 weeks all wounds were healed. His strength gradually increased, as well as the use of his left arm. He gained in weight and his color improved remarkably. Appetite was good and on discharge, 6-18-33, he was looking quite healthy, much better than he had for many months previously. His only complaint was some dyspnea on exertion and this was not marked. His respiratory function improved much after his heart had become compensated to his being up and about. He had been walking about for 2 weeks at time of discharge from hospital. Vital capacity on admission 3,500; at discharge 1,650. At time of discharge his blood showed 5,100,000 RBC., 8,500 WBC., and 90% HB. On discharge, his ECG. was practically normal.

Weight on admission 4-4-33 145 lbs.. On May 21, 1933, his weight was 130 lbs.. On June 14, 121½ lbs., June 18, 122½ lbs., June 28, 126 lbs..

Operative Report

Anesthetic Remarks: Uneventful. Pulse good volume. Color good. Glucose given when B.P. fell. Pt. responded at completion of operation. Blood transfusion was given.
Dictation: Dr. Graham.

An incision was made over the 6th rib; the rib was removed from the transverse process to the anterior axillary line. The 7th rib was also removed in the same way and the intercostal bundle was excised. The pleura was opened and the upper lobe was found to be atelectatic. Several masses could be felt within it suggestive of infiltration with carcinoma. Although the apex of the lung was free from adhesions there were many other adhesions between the lung and the chest wall. There were particularly dense ashesions between the upper lobe and pericardium, and also posteriorly between the upper lobe and the parietal pleura. The lower lobe was adherent everywhere to the chest wall. In the upper part of the upper lobe several firm nodules were felt which were suggestive of carcinomatous metastases. In attempting to separate the upper lobe from the lower lobe, it was found that the interlobar fissure was not fully developed. There were also some nodules in the upper part of the lower lobe which were suspicious of carcinomatous involvement. It was felt that not only would it be very difficult to remove the upper lobe alone, but also that in doing so some of the cancer would be left behind. Consequently it was decided to remove the entire left lung. After separating the adhesions, most of which required clamping and cutting followed by ligation, the pedicle was freed and a small catheter was tightly secured around it. It seemed preferable to use the soft pressure of a rubber catheter rather than a crushing clamp for this purpose, in order to preserve the blood supply as much as possible to the end of the stump of the bronchus. The idea behind this was to encourage healing of the bronchial stump as much as possible. Distal to the catheter two clamps were placed on the entire pedicle and an incision was made between them thereby cutting away the entire lung. After cutting away the lung, the open stump of the bronchus was cauterized thoroughly with the actual cautery and then swabbed with 25% silver nitrate solution. The stump was then transfixed with a needle carrying a double thread of #2 chromic catgut. This was tied securely around the whole pedicle. Another double ligature of #2 chromic catgut was placed in a position slightly distal to the first ligature, and finally a third ligature of the same sort was applied. The catheter was then removed and no bleeding from the stump occured. The open end of the stump of the bronchus was slightly less than one inch from the bifurcation of the trachea. The aorta was plainly visible immediately posterior to the stump of the left bronchus. Because the entire lung had been removed there was no tissue available for covering over the stump of the bronchus. Two enlarged mediastinal glands, which could be seen immediately below the bifurcation of the trachea, were removed for microscopic examination. These, however, seemed soft and evidently did not contain any cancer. Because the patient's condition seemed excellent, and because it was felt desirable to obliterate the pleural space as much as possible, additional ribs were removed

from the transverse processes to the anterior axillary line. The ribs removed were the 4th, 5th, 6th, 7th, 8th, 9th and 10th. Thru a small stab wound a small catheter was inserted below the line of incision and carried into the pleura cavity just below the stump of the pedicle. This catheter fitted tightly so that no leakage of air at all occured around it. The wound was then closed in layers and it was noted that the soft tissues collapsed readily into the pleural cavity. No attempt was made to suture the parietal pleura together. The 6th and 7th muscle bundles had been removed in order to give exposure, but the rest of the muscle bundles were not cut away. The patient was given a blood transfusion in the operating room and left the operating room with blood pressure and pulse the same as they had been at the beginning of the operation. The rubber catheter was connected with a longer rubber tube which was brought down to the level of some boric acid solution in a bottle in order to give air tight drainage.

SURGICAL PATHOLOGY BARNES HOSPITAL

Name	Gilmore, Dr. J.L.	Register No.	89475	Age	48
Address		Ward		Date	4-5-33 (11-11-48)
Surgeon	Dr. Graham	Specimen No.	21246		

Gross Pathology: (11-11-48) The gross specimen consists of the entire left lung which is received in Kaiserling fixative. The entire lower section through the entire lung at 2 to 3 cm. intervals has been made. There is a tumor mass ulcerating into the upper lobe bronchus near the hilum which on cut section appears to be well delineated and measures 3 cm. in its greatest diameter. Distal to this tumor in the upper lobe there are numerous pinpoint abscesses in the lung parenchyma. Over the posterolateral apical region of the upper lobe there is a markedly thickened pleura which appears to have a portion of the parietal pleura attached to it. The lower lobe appears to be essentially normal without abscesses, obstruction or abnormalities of the pleural surfaces. On gross examination the superior interlobar bronchial node which is immediately adjacent to the tumor appears to be directly involved in the process. There are several other nodes about the upper lobe bronchus which do not appear to be involved in the tumor process. Several small nodes about the lower lobe bronchus are likewise negative grossly. Many black and white and colored photographs were taken of the fixed gross specimen. The following additional sections are taken.

A_X—a section of the tumor to show its entire extent
A—4 to 5 additional sections of the tumor
B_1—6 lymph nodes from about the upper lobe bronchus
B_2—a section from the thickened pleural surface at the posterior apex of the upper lobe
D_1—3 nodes from about the lower lobe bronchus Jar IV (Black)

Microscopic Pathology: The surface of the tumor as it projected into the bronchus showed a fairly well differentiated pattern with numerous epithelial pearls. The tumor extended into the lung parenchyma destroying bronchial cartilage and invading contiguous lymph nodes. In the deeper areas of the neoplasm it was extremely undifferentiated with practically no keratinization and with numerous mitotic figures. In one area of the neoplasm, as it arose from the bronchus, there was squamous metaplasia and epidermoid carcinoma in situ. Further nodes, 11 in number, showed no evidence of tumor.

Diagnosis: Lung, bronchus, left, upper — Epidermoid carcinoma, grade III
Lymph nodes, regional — Epidermoid carcinoma, grade III, invasive
Lymph nodes, regional — Hyperplasia, 11/11
Lung — Bronchial pulmonary suppuration with multiple small abscesses

Lauren V. Ackerman, M.D.

It is important to note that the review of the tissue by Dr. Lauren Ackerman in 1948 established that the lesion was an invasive epidermoid carcinoma. Moreover, there was involvement of a lymph node.

Two postoperative notes by our distinguished colleague, Dr. William Adams, then a Fellow in Surgery, are worth recording.

4-6-33 3:15 Adams "Patient quite comfortable unless he moves. He had no M.S. since 8 A.M. Has not voided since operation. B.P. remaining at 120/ No cyanosis or dyspnea. Pulse steady. Drained about 800 cc through catheter during interval from 12 M yesterday until 11 A.M. today. Little drainage since. Temp. down to 37.2° this noon. O_2 tank to be kept in room and O_2 tent available in case of emergency."

4-18-33 Adams "Patient's temp. has risen to 39.7° tonight. Pulse 134. R 32. No cyanosis. States he is having some respiratory difficulty. Coughing if attempts to lie on rt. side or on talking. Dr. Graham notified of condition. Given Aspirin 10 gr. and Codeine 1 gr. to lower temp."

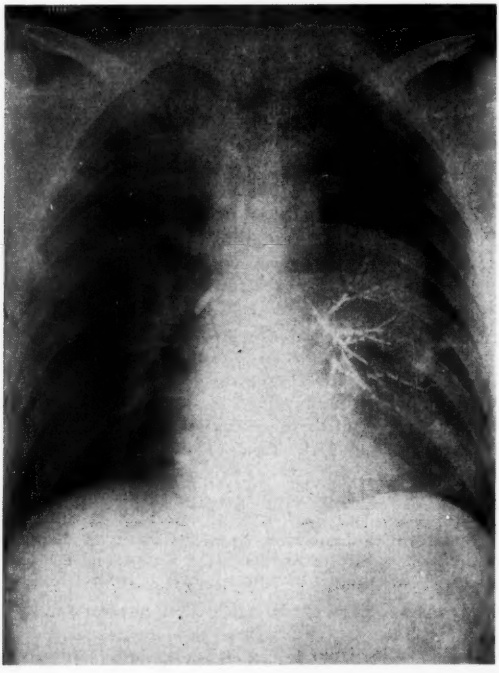

Fig. 1.—Roentgenogram showing pneumothorax and Lipiodol bronchogram. The pneumothorax was accidentally induced when a thoracentesis was performed.

4-19-33 Adams "Patient's general condition about the same this A.M. except less respiratory distress, less cough, and less pain since midnight. Fluoroscopy reveals similar condition as that seen yesterday morning. Under general anesthesia a #14 catheter was inserted through a trocar into cavity at level of 4th dor. vert. just medial to scapula. 150 cc of thin redish-brown pus was removed. Open drainage established."

It appears the bronchus began to leak 13 days after the operation. Certainly no one could accuse the staff of prescribing too much medication.

A few years after the first successful pneumonectomy for carcinoma of the lung, rumors were circulated that the lesion was not really a carcinoma, but

an adenoma. The amazing longevity of misinformation has been demonstrated because to this day one occasionally hears this misstatement repeated. It is appropriate, therefore, to show a photograph of the gross specimen and the photomicrograph. (Figs. 1, 2, and 3).

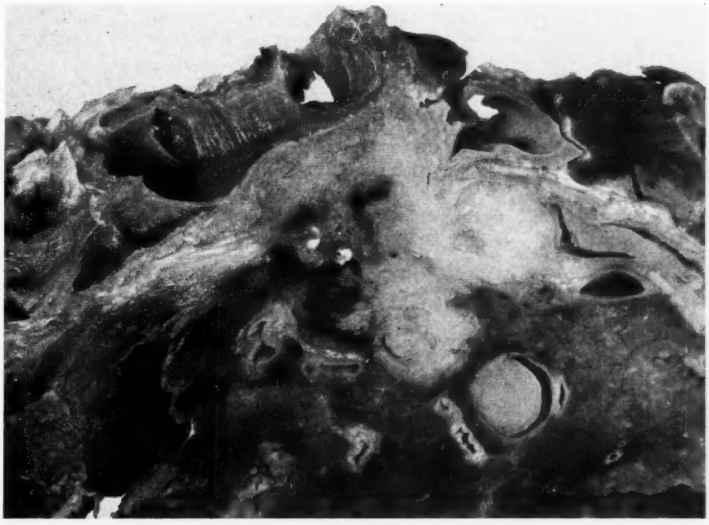

Fig. 2.—The gross specimen showing a bronchogenic carcinoma in the left upper lobe.

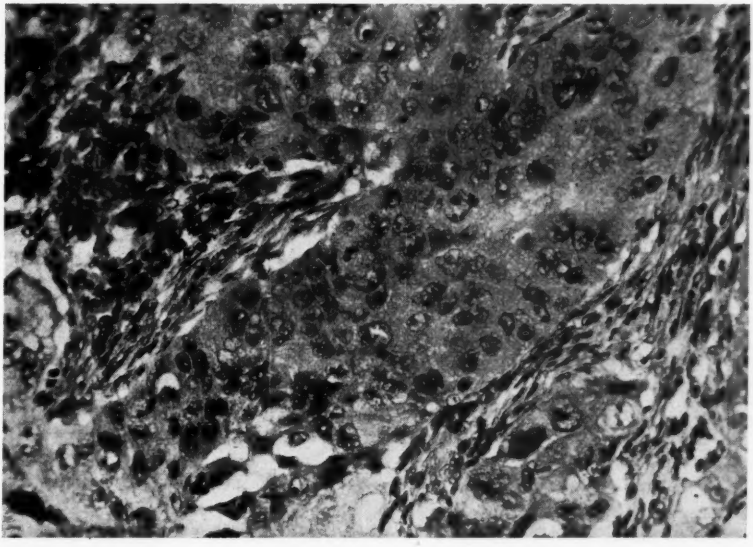

Fig. 3.—Microscopic section showing undifferentiated carcinoma.

While the patient was recovering from the pneumonectomy, thoracoplasties, and thoracostomies, the meeting of The American Association for Thoracic Surgery was held on May 9, 10, and 11, 1933. Before attending in absentia the Sixteenth Annual Meeting of the Association in Washington, D. C. it is important to conclude the case report. The patient is vigorous, living and well twenty-five years after the removal of the left lung.

THE AMERICAN ASSOCIATION FOR THORACIC SURGERY

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The American Association for Thoracic Surgery
Washington, D. C., May 9, 10, 11, 1933
Hotel Headquarters — The Willard Hotel

Program

Tuesday, May 9

9:00 A.M. National Museum Building.

1. Business Meeting of the Association.
2. The Results of Phrenic Nerve Operations in 225 Cases With a Discussion of the Technique of the Operations.
   Dr. H. Ryerson Decker, Pittsburgh.
3. An Estimate of the Value of Phrenic Nerve Interruption for Phthisis Based on 654 Cases.
   Dr. John Alexander and Dr. Lawrence Nehil, Ann Arbor.
4. Experimental Study of the Effect of Phrenicectomy on Cough.
   Dr. Herbert A. Carlson, Minneapolis, Dr. Harry C. Ballon, Montreal, Dr. Hugh M. Wilson and Dr. Evarts A. Graham, St. Louis.
5. Thoracoplasty.
   Dr. E. J. O'Brien, Detroit.
6. The Operative Mortality From Thoracoplasty in Pulmonary Tuberculosis. Analysis of 6 Fatal Cases.
   Dr. H. L. Beye, Iowa City.
7. A Study of 150 Cases of Thoracoplasty for Pulmonary Tuberculosis During the Past Two Years — Reflection of Technique and Results.
   Dr. Pol. N. Coryllos, New York City.
8. Bronchogenic Carcinoma. Special Reference to Its Classification, Prognosis and Treatment.
   Dr. Louis H. Clerf and Dr. B. L. Crawford, Philadelphia.

1:00 P.M.

1. Collapse Therapy and the Ambulatory Patient.
   Dr. J. A. Myers, Minneapolis.

2. The Future Surgical Status of the Collapse Therapy Patient.
   Dr. T. J. Kinsella, Oak Terrace, Minn.
3. Pneumocavernolysis in the Treatment of Pulmonary Tuberculosis With Cavitation.
   Dr. Harold Neuhof, New York City.
4. Non-Penetrating Wounds of the Heart. The Cardiac Manifestations Brought About by Acute Peripheral Compression.
   Dr. Claude S. Beck and Dr. Ernest Bright, Cleveland.
5. Removal of Needle From the Heart With Electrocardiographic Studies Before, During, and After Operation.
   Dr. Francis Scrimger, Montreal.
6. The Diagnosis and Treatment of Encapsulated Empyemata.
   Dr. W. A. Hudson, Detroit.
7. Moving picture study of the treatment of acute empyema.
   Dr. Jos. Gale, Madison.
8. Moving picture study of the treatment of acute empyema.
   Dr. Ralph B. Bettman, Chicago.

Wednesday, May 10

9:00 A.M.

1. Executive Meeting of the Association.
2. President's Address.
   Dr. George P. Muller, Philadelphia.
3. Address on Lung Abscess and Pleural Effusion.
   Dr. Marc Iselin, Paris France. (By special invitation.)
4. Pneumonectomy for Sarcoma of Lung in a Tuberculous Patient.
   Dr. Howard Lilienthal, New York City.
5. Dermoids of the Mediastinum.
   Dr. Carl A. Hedblom, Chicago.
6. Intrathoracic Teratomas.
   Dr. S. W. Harrington, Rochester, Minn.
7. Esophagectomy for Cancer of the Upper Esophagus With Lessons Derived From an Operative Failure.
   Dr. P. E. Truesdale, Fall River, Mass.
8. Experiences With Oleothorax Treatment.
   Dr. John N. Hayes and Dr. Lawrason Brown, Saranac Lake.

1:00 P.M.

1. Active Contractility of the Bronchopulmonary Smooth Muscle as Demonstrated by Electrobronchographic Records.
   Dr. Ethan F. Butler, Elmira, N. Y.
2. An Experimental Study of the Reactions of the Pleura to the Bacillus of Tuberculosis.
   Dr. W. S. Lemon, Rochester, Minn.
3. Effects of Laparotomy and Abdominal Distention on the Lung Volume.
   Dr. Edward D. Churchill, Dr. H. Beecher, Dr. H. H. Bradshaw, and Dr. G. E. Lindskog, Boston.
4. The Treatment of Traumatic Haemothorax. An Experimental and Clinical Study.
   Dr. D. C. Elkin and Dr. J. C. Sandison, Atlanta.
5. Physiological Determinants in Surgery for Relief of Cardiac Pain.
   Dr. Peter Heinbecker, St. Louis.
6. The Tensile Strength of the Paralyzed Diaphragm.
   Dr. Richard H. Meade, Jr., Philadelphia.
7. Extreme Compression and Cirrhosis of the Lung. A Preliminary Experimental Study.
   Dr. C. M. Van Allen, Peiping, China.

8.00 P.M. PRESIDENT'S BANQUET.

Thursday, May 11

9:00 A.M.

1. Bronchiectasis.
   Dr. James A. Miller, New York City.
2. Selective Surgical Collapse for Lung Abscess.
   Dr. Richard H. Overholt, Boston.
3. Fixation of Chest Lesions With Subsequent Compression.
   Dr. W. P. Herbert, Asheville.
4. Oidiomycosis of the Lungs.
   Dr. David T. Smith, Durham, N. C.
5. Experimental Pulmonary Lesions of Aspergillus Niger: Superimposition of This Fungus on Experimental Pulmonary Tuberculosis.
   Dr. Norman Bethune, Montreal.
6. Suture of the Lung.
   Dr. Frank S. Dolley, Los Angeles.
7. Intrapleural Pneumolysis. A Critical Review.
   Dr. Julian A. Moore, Asheville.

The following officers were elected for 1934:

President	Dr. George J. Heuer	New York City.
Vice President	Dr. John Alexander	Ann Arbor, Mich.
Secretary	Dr. Duff S. Allen	St. Louis, Mo.
Treasurer	Dr. Edward D. Churchill	Boston, Mass.
Editor	Dr. Evarts A. Graham	St. Louis, Mo.

The following were advanced from associate to active membership:

Dr. I. A. Bigger	Charlottesville, Va.
Dr. Alfred Blalock	Nashville, Tenn.
Dr. Peter Heinbecker	St. Louis, Mo.
Dr. W. A. Hudson	Detroit, Mich.
Dr. Richmond L. Moore	Asheville, N. C.
Dr. LeRoy S. Peters	Albuquerque, N. M.
Dr. David T. Smith	Durham, N. C.
Dr. Owen H. Wangensteen	Minneapolis, Minn.

The following were elected to associate membership:

Dr. William Adams	Chicago, Ill.
Dr. Clyde Allen	Detroit, Mich.
Dr. J. B. Amberson	New York City
Dr. Marr Bisaillon	Portland, Oregon
Dr. Maurice Fisher	Rochester, N. Y.
Dr. Wallace Frank	Louisville, Ky.
Dr. Cameron Haight	Ann Arbor, Mich.
Dr. George F. Herben	Loomis, N. Y.
Dr. Chevalier Jackson, Jr.	Philadelphia, Pa.
Dr. Foster Murray	Brooklyn, N. Y.
Dr. J. W. Nixon	San Antonio, Texas
Dr. George Ornstein	New York, N. Y.
Dr. John Powers	Boston, Mass.
Dr. Leo Rigler	Minneapolis, Minn.
Dr. Willard Van Hazel	Chicago, Ill.

Of the thirty-five scientific papers presented twenty-five years ago more than a third were on various aspects of the treatment of tuberculosis. Interest in the heart was manifested by three important papers. There was only one presentation concerning bronchogenic carcinoma and two on mediastinal tumors. The subjects of empyema and lung abscess had their share of attention and it appears that one half day was devoted to experimental studies in much the same way that time is set aside for the forum in our present programs.

The esophagus rated only one paper with lessons reported from a failure of esophagectomy for carcinoma of the upper third of the esophagus. History has a strange way of repeating itself.

There were sixty-seven members present at the meeting twenty-five years ago, fifty-three active, twelve associate, and two senior members; a sharp contrast to the Thirty-Seventh Annual Meeting of the Association in Chicago at which there were more than one thousand registrations.

The meeting in Washington was held in a small lecture room of the Smithsonian Museum. The place of the meeting was determined by the Congress of American Physicians and Surgeons. This was the last time The American Association for Thoracic Surgery met as a component member of the Congress.*

The Program Committee this year received one hundred and fifty-four abstracts for consideration. Enough outstanding papers were available to fill three full programs. One might long for the good old days, but a quarter of a century ago the trials of the program committee were expressed by Dr. Muller in his presidential address.

> "This year for the first time your Program Committee has been embarrassed by the quantity of material offered and found it difficult to follow the rule of accepting all offerings from members. It seems to me that in the future, committees will find it expedient to select those papers which offer new thoughts or are sufficiently controversial to excite discussion."

One paper deserves special attention. Not for the presentation itself, but because of the battle of semantics which it precipitated. I refer to the paper "Pneumonectomy for Sarcoma of the Lung in a Tuberculous Patient" by Dr. Howard Lilienthal.

It is safe to assume that Dr. Graham was elated about the patient back in St. Louis who had survived the removal of an entire lung. It is not surprising, therefore, for him to discuss Dr. Lilienthal's pneumonectomy case in a confident way and to describe his now convalescent patient and the details of the case in a scholarly manner. The trespass into the field of etymology, however, was most unfortunate.

A portion of Dr. Graham's discussion is as follows:

> Dr. Evarts A. Graham (St. Louis).—I was much interested in this case of Dr. Lilienthal because he operated upon his case just a short time before I performed a complete

*By authority of Dr. Ethan Flagg Butler's "little black book." Dr. Duff Allen, the Secretary could not be present because Mrs. Allen was having a baby. Dr. Butler was drafted to serve as secretary.

pneumectomy. In my case, however, fortunately the result was successful. I do not call it pneumonectomy, as Dr. Lilienthal does, because I have the support of the Oxford dictionary and various other dictionaries to call it pneumectomy instead of pneumonectomy.

The Oxford dictionary and other references did not impress Dr. Pol. Coryllos who not only was a Greek, but knew the Greek language well.

Dr. Pol. N. Coryllos (New York).—(Illustrating on blackboard.) First a point of etymology. I agree with Dr. Lilienthal that the correct Greek term for excision of the lung is "pneumonectomy" and not "pneumectomy" as proposed by Dr. Graham. The lung in Greek is πνεύμων-ονος, whereas πνεῦμα-ατος means air or spirit. The second part of the term, "εχτομὴ" (excision) indicates the action exerted upon the lung, therefore the latter must be placed in the genitive (πνεύμονος) the radical of which is πνεύμον- so that the composed word will be "pneumonectomy." "Pneumectomy" on the contrary means "resection of air," and if correctly constructed should be pneumatectomy. It is according to the same rules that are constructed the terms gastrectomy, ureterostomy, salpingectomy, otorhinolaryngology, etc. I hope that this literary digression will help to settle that question.

This must have been a bitter pill since Dr. Graham was a real stickler on diction, grammar, and particularly superior about his knowledge of word roots.

It is impossible to determine accurately the exact number of surgeons who were engaged in a significant amount of thoracic surgery twenty-five years ago. Certainly no more than a hundred in the United States and Canada, probably considerably less. Only a few limited their work to the chest and most of them were prominent general surgeons who were interested in developing a new field. There were a few physicians with little or no surgical training attempting to do chest wall operations on patients in the tuberculosis sanatoriums.

Opportunities for training in thoracic surgery were limited. In most instances the program was integrated with general surgery with sometimes the addition of fellows who might spend an extra year or two in clinical work and research projects connected with thoracic surgery. A few exceptions, however, were developing. In January of 1933, Dr. John Alexander established at the University of Michigan a training program in thoracic surgery with a minimum appointment of two years' duration. Others, including Dr. E. J. O'Brien, Dr. Pol. Coryllos, and Dr. Louis Davidson established residencies devoted exclusively to surgery of the chest.

Two philosophies were beginning to develop. One, a concept of exclusive specialization in thoracic surgery. The second and more popular then, additional training for the surgeon who might also operate in the chest, but remain principally in general surgery.

Twenty-five years ago only a small minority accepted the feasibility of specialization. The field seemed too small; the patients were poor physical and financial risks; and economic suicide was often predicted for those foolish enough to desert general surgery.

Reliable data concerning the number of surgeons in this country limiting their practice to thoracic surgery are not available. It has been estimated that in the neighborhood of two hundred and fifty do so. The remainder of those

now certified by the Board of Thoracic Surgery do not. After twenty-five years the differences of opinion concerning specialization remain, but in different proportions.

It is apparent, however, that the dire financial disasters predicted for the specialists were unwarranted. An important factor, of course, has been the thoracic surgeon's successful wrestling match with nose and throat specialists for the control of endoscopes.

Originally the members of our Association were not receptive to establishing a board of Thoracic Surgery. It was first discussed in 1936 and a committee appointed to study the question. In 1937 the committee's opinion was that there was no need for certification of thoracic surgeons by a separate Board at that time.

In 1945, a second committee was appointed and made its report in 1946. A recommendation was adopted favoring the formation of a Board of Thoracic Surgery as a subsidiary of the American Board of Surgery. A plan of organization was established in 1948 by the Eggers Committee. And the Board of Thoracic Surgery was incorporated under the laws of the State of Michigan on Aug. 31, 1950, as a subsidiary of the American Board of Surgery.

There are now two hundred and twenty-eight founder members, and in the decade of existence five hundred and thirty-four candidates have been certified by examination.

A total of seven hundred and sixty-two surgeons have a certificate stating they are properly qualified to operate upon the chest of their fellow man. The recent crop of thoracic surgeons has been abundant. Two hundred and fifty-nine have been certified in the past 3 years. It might appear that the principle of supply and demand is being violated.

The establishment of first the American Board of Surgery and later its subsidiary, the Board of Thoracic Surgery, stimulated surgical segregation. Influences on rank and assignment during World War II gave a final and mighty emphasis to Board certification.

Practically all surgical specialties were originally developed by general surgeons, but in most instances have become distinct and separate fields of endeavor as the specialty develops.

Incredibly complete severance with one of the oldest surgical specialties has been so radical that surgeons certified by other Boards must relinquish the previously conferred sheepskin to become eligible for certification.

Fortunately, there is little chance of surgical contamination in this particular specialty since only one year of internship is required before the special training.

The tendency to drift from old patterns of emphasis on general surgery is shown by the requirements of other Boards. For example, the Neurosurgical Board requires only one year of surgical internship preliminary to training in the specialty. The Board of Orthopedic Surgery specifies a year of internship and one year of general surgical residency as a preliminary.

The Board of Thoracic Surgery is a subsidiary of the American Board of Surgery. Total dependence on general surgery is established if one examines

the definition of subsidiary—meaning: furnishing aid, auxiliary, tributary, as a capacity; secondary.

No Board has more rigid requirements. Certification by the American Board of Surgery and an additional two years of training.

Our rigid standards resulted from the philosophy that all engaged in thoracic surgery must first be trained in general surgery. Certainly one of the reasons for this particular emphasis was to discourage physicians who entered the field via the chest wall route without previous experience in surgery. Time and the combination of antibiotics with the modern surgical treatment of tuberculosis have practically eliminated this group. Probably another factor was the failure of other specialty Boards to require adequate basic surgical training.

Fortunately the original plan of two written and two oral examinations has been condensed with the elimination of the written examination in thoracic surgery. The first part of the general surgical Board, however, now includes a certain prescribed percentage of questions relating to thoracic surgery. This change, however, has created problems for the young surgeon who has little or no contact with thoracic surgery during the general surgical training.

It is easy to comment that in a properly integrated service the trainee should have sufficient background to answer correctly the questions on thoracic surgery. In many instances, however, this is simply not the case. Depending upon the pattern of training the general major and the thoracic major might find this examination quite difficult.

There are now eighty-nine hospitals approved for training in thoracic surgery. One wonders if there are really eighty-nine institutions in existence in our country with sufficiently varied clinical material and available teachers. This is a particularly pertinent question in regard to surgery of the heart and great vessels.

Neither the American Board of Surgery nor the Board of Thoracic Surgery attempt to define independently an acceptable training program. Residencies in surgery are judged by a conference committee consisting of representatives of the Council of Medical Education of the American Medical Association, the American College of Surgeons, and the American Board of Surgery. Moreover, the American Board of Surgery and its subsidiary, the Thoracic Board have made it clear that they are not concerned with special privileges or recognition their diplomates may obtain by virtue of certification. This is in sharp contrast to many specialty groups who make their own ground rules for training programs and emoluments for their certificates.

Thoracic surgery is now a distinct specialty and it is safe to predict it will become even more so. And, it makes little difference how each of us in this Association feels about it. The public and the nonsurgical segment of the medical profession have nurtured specialists and, in fact, demand them.

Otherwise intelligent members of our profession are very apt to classify a colleague as a Board man or not a Board man. The layman has a similar

approach with different terminology. The surgeon is a heart man, lung man, or some kind of man whom they associate with the repair or eradication of various vital or semivital organs.

It would be difficult for surgeons who have fulfilled the qualifications for certification in thoracic surgery to lose interest in general surgery. Once, however, they are labeled they are often confined to a single field of endeavor. This is particularly true of those in private practice. A small minority of surgeons in full time clinics and medical schools are not quite so limited, but even in these institutions the trend is toward regional specializaton as far as operating is concerned.

Although the battle for various organs continues, the chest man has been gaining ground, or perhaps better stated, not infrequently successfully raids outside of his designated body cavity.

On the left he can slip through the diaphragm for spleens or stomachs without treaty violation serious enough to cause a major battle. And on the other front, a thyroid gland which isn't well above the sternal notch is fair game.

Conversely, surgeons now designated as generalists are fighting valiantly on two fronts. They have been pushed down on the left flank by the chest specialist and forced upward on the other major battle-field by the colon-rectal specialist who also has been commissioned by his Board to engage in this war. The war with the neurosurgeons was lost years ago. For the most part bones have been taken away by the orthopedist, and even burns by the plastic surgeon. The general surgeon still controls the right flank of liver and gall bladder and the middleground, but his supply lines are thin.

This half facetious, but not far from the truth, description of the state of surgical affairs may create serious problems for the young surgeon.

In the five to seven years after graduation from medical school this now not so young surgeon, trained in general and thoracic surgery, leaves the institutional nest—not because he is incompetent or not wanted—the nest is full, and there is the possibility he has had about all of the nest he can tolerate. He searches and finds a community he admires and in which there is a reasonable chance to make a living. He may then find it is expedient or totally necessary to pattern his practice by default.

If in the community there are three well-established, fairly young thoracic surgeons and the majority of the general surgeons are senile or well on the way—general it will be, or the converse may be true. In either event there has been a waste of talent, energy, and money.

Prerequisite training in general surgery before specialization I sincerely hope will continue. And it is proper to suggest that other specialties would profit by following this example.

It appears, however, present standards or, better stated, rules for training are not realistic and perhaps untenable.

For example, is it really necessary in every case to spend four years following internship in general surgery before training in thoracic surgery? Is it reasonable to prescribe a six months' minimum credit for the study in basic

sciences? For the interested student the study of physiology, chemistry, or almost any science might result in a potential contributor to the progress of surgery. Conversely, it would be utterly ridiculous to require all, including the uninterested to follow the same pattern of postgraduate education.

These questions are easy to ask, but extremely difficult to answer in a specific way. It is suggested, however, there be fewer rules and that more attention be given to quality of training rather than quantity in terms of years.

A quarter of a century has passed since the pneumonectomy of our case report was performed by the man whose memory we honor today. During this time, thoracic surgery has progressed from a faltering, dangerous formative stage to a recognized surgical specialty.

More than any other organization in the world our Association will influence the trends of the next twenty-five years. It is appropriate then, to ask ourselves just what have we done and more important, what are we going to do.

Thanks are expressed to Remington Rand, Inc., St. Louis, for providing photostats of the original records which had been placed on microfilm.

I am deeply indebted to Doctors Ackerman, Bradley, Burford, and Moyer who made records and the photographs of the specimen available, and to Dr. James Gilmore who gave permission to review the case report.

FURTHER STUDIES IN THE SURGICAL MANAGEMENT OF CARCINOMA OF THE LUNG

A Further Study of the Cases Treated at the Massachusetts General Hospital From 1950 to 1957

Edward D. Churchill, M.D., Richard H. Sweet, M.D., J. Gordon Scannell, M.D., and Earle W. Wilkins, Jr., M.D. (by invitation), Boston, Mass.

EIGHT years have elapsed since the previous report of the surgical management of primary carcinoma of the lung at the Massachusetts General Hospital. That report[1] included all cases seen in the years 1930 to 1950 with follow-up data on all resections carried out prior to January, 1948. The present study brings up to date the results of surgical management of cancer of the lung at the same hospital by adding all cases seen in the period January, 1950, to January, 1957, with follow-up information on *all* resections performed before January, 1956. The follow-up data have been divided into 2 groups identified as "present series" which includes all resections carried out between January, 1948, and January, 1956, and "original series." The latter includes all cases of resections prior to 1948. The two groups are considered together as a "total" series of 341 cases followed to January, 1958, representing all patients undergoing resection for carcinoma of the lung at the Massachusetts General Hospital from 1933 to 1956. Follow-up data are available on all patients.

The present study, as the previous one, includes all patients admitted to the general hospital wards as well as those admitted to the private divisions. Since the operations have been carried out by a great number of individuals on both the visiting and resident staffs, it would be unwarranted to assume a standardized technique or extent of resection. In general, the principles of operation were those outlined in the previous report. Lobectomy, with removal of all gross evidence of disease, was considered reasonable when there was evidence of diminished pulmonary or cardiac reserve, when uncertainty of the diagnosis existed and the lesion could be totally excised by lobectomy, when the lesion was small, peripheral and without evidence of lymph node

From the General Surgical Services of the Massachusetts General Hospital and the Department of Surgery of the Harvard Medical School, Boston, Mass.

Read at the Thirty-eighth Annual Meeting of The American Association for Thoracic Surgery at Boston, Mass., May 16-18, 1958.

extension, and when the carcinoma had extended beyond surgical bounds but the bulk of the primary tumor could be removed by lobectomy with the hope of relieving symptoms. Again, it should be emphasized that the majority of lobectomies were carried out not merely as a local excision of a primary growth, but as a complete dissection of an anatomic unit of the lung to include the adjacent lymph nodes. Similarly, pneumonectomy included as extensive dissection of adjacent nodes as the operator deemed advisable, with or without intrapericardial division of the hilar structures as the situation demanded. In the present study no effort has been made to separate "radical" as opposed to "simple" pneumonectomy or lobectomy nor to correct for those resections undertaken when it may have been the estimate of the surgeon that the disease had extended beyond the limits of extirpation.

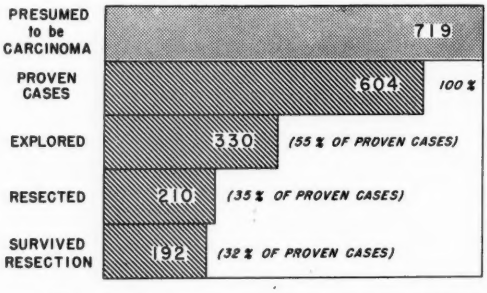

Fig. 1.—Primary carcinoma of the lung, 1950 to 1957.

Specifically excluded are all cases of bronchial adenoma, either the carcinoid or cylindroma types, all carcinomas that might reasonably be considered metastatic, and all cases of pulmonary alveolar adenomatosis (alveolar cell or bronchiolar carcinoma). Although the latter form an extremely interesting group of cases, their nature is sufficiently debatable to warrant exclusion from the group commonly termed bronchogenic carcinoma.

Between Jan. 1, 1950, and Jan. 1, 1957, a clinical diagnosis of carcinoma of the lung was entered in the records of 719 patients (Fig. 1). In 604 of these, the clinical diagnosis was verified by histologic means. Of the 604 proved cases, 330 or 55 per cent came to thoracotomy. In 210 of these (35 per cent of the proved, 64 per cent of the explored), resection of the tumor was possible by pneumonectomy or lobectomy. One hundred and ninety-two patients survived resection and left the hospital alive (32 per cent of the proved cases). Since the 5-year cumulative survival rate for this period was approximately 30 per cent, it can be assumed that 63 patients survived or could be expected to survive 5 years (10.4 per cent of the proved cases). When compared to our earlier report these figures represent some improvement. The corresponding percentages in the 1930-1950 patients are: proved cases 681, explored 294 (43 per cent); resected 171 (25 per cent of proved); survived resection 137 (20 per cent).

It is hoped, but by no means certain, that the increase in the number of resectable tumors reflects an increasing awareness by the medical community of the constructive role of surgery in the management of carcinoma of the lung and the value of early diagnosis. In type and duration of symptoms the patients of the present series differ very little from the former series or from the many other large series recorded in the literature. There is, however, one new and clinically identifiable group, namely, the "survey" case, a designation we have applied to the individual whose pulmonary cancer was discovered as a result of investigation initiated by the presence of a suspicious shadow in a chest x-ray film taken in the course of a survey, routine physical examination, or study of an apparently unrelated medical or surgical problem.

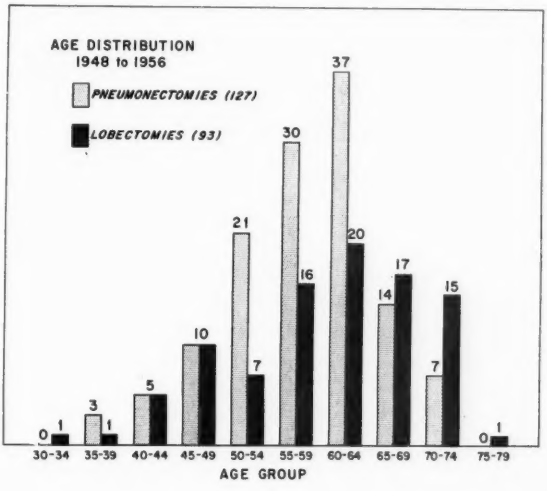

Fig. 2.

Age distribution (Fig. 2) has shown no marked change, although a greater number of older patients come to operation now than formerly. The increased age of the patients may account in part for the increased percentage of lobectomies in the present compared to the earlier report. In the period 1948 to 1956, there were 93 lobectomies and 127 pneumonectomies (43 per cent lobectomies). In the period 1933 to 1948, there were 34 lobectomies, 87 pneumonectomies (28 per cent lobectomies).

Operative mortality for pulmonary resection appears to have stabilized at a reasonable level. Among 127 pneumonectomies in the years 1948 to 1956, there were 13 hospital deaths—a mortality rate of 10 per cent. In the same years, there were 93 lobectomies with 6 hospital deaths—a mortality rate of 6.4 per cent. It is interesting in view of the widely held clinical impression that right pneumonectomy is a more severe operation than left that, in this same period, there were 66 right pneumonectomies with 6 deaths (9 per cent)

and 61 left pneumonectomies with 7 deaths (11 per cent). That right pneumonectomy was avoided in favor of lobectomy which carried an over-all mortality of 6.4 per cent is suggested by the fact there were 58 right-sided as opposed to 35 left-sided lobectomies in the same period.

SURVIVAL

The survival of patients undergoing resection is presented in the form of cumulative survival rates similar to life tables. The method has been described in detail by Hill[2] and by Puffer.[3] Table I is representative of the method. Here

TABLE I. CUMULATIVE SURVIVAL
1948-1956

TIME PERIOD ENDING (YEARS)	DEATHS	DROPPED FROM STUDY	PATIENTS ENTERING	PATIENTS AT RISK	PERIOD MORTALITY	PERIOD SURVIVAL	CUMULATIVE SURVIVAL
			Pneumonectomy				
½	43	0	127	127	34	66	66
1	17	0	84	84	20.2	79.8	53
1½	14	0	67	67	20.9	79.1	42
2	6	0	53	53	11.3	88.7	37
2½	5	1	47	46.5	10.8	89.2	33
3	6	1	41	40.5	14.8	85.2	28
3½	3	1	34	33.5	8.9	91.1	25
4	0	1	30	29.5	0	100	25
4½	0	2	29	28	0	100	25
5	1	1	27	26.5	3.8	96.2	24
			Lobectomy				
½	20	0	93	93	21.5	78.5	79
1	11	0	73	73	15.1	84.9	67
1½	6	0	62	62	9.7	90.3	60
2	4	0	56	56	7.1	92.9	56
2½	6	1	52	51.5	11.6	88.4	50
3	8	2	45	44	18.2	81.8	41
3½	2	2	35	34	5.9	94.1	39
4	3	0	31	31	9.7	90.3	35
4½	1	3	28	26.5	3.8	96.2	34
5	1	2	24	23	4.3	95.7	33

are presented the cumulative survival rates for all patients undergoing pneumonectomy in the years 1948 to 1956 followed to January, 1958. Date of operation is taken as zero time for a given individual. Mortality rates are calculated for each 6-month period thereafter by dividing [the number of individuals entering a given period and at risk during that period] by [the number of deaths occurring during that period]. If the mortality rate for a given period is subtracted from 100 per cent, a survival rate for the period results. If this, in turn, is multiplied by the cumulative survival rate for the previous period, a cumulative survival rate for the entire series at a given time period is established. The results can be effectively shown graphically (Fig. 3). The method permits the maximum use of a group of patients under study by correcting for patients who are lost to follow-up or for other reasons drop out of the study. In the present material, no patient was lost to follow-up except by virtue of the

fact that operation was done an insufficient length of time prior to the time period under study. Thus, a patient operated upon 26 months before his last recorded follow-up note would drop out of the study after the time period ending at 2 years, and he would appear in the column marked "lost to the study."

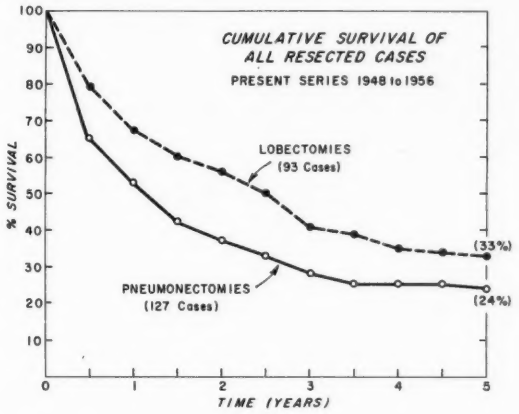

Fig. 3.

By convention, such an individual would be considered "at risk" during half the time period in which he was lost to follow-up. The number of "patients at risk" during a given period, therefore, is the number entering a given period minus one half the numbers of patients "lost" to the study during that period. In order to permit comparison and consolidation the survival data of the previous report have been recalculated according to this method of Hill. Table II presents the 10-year cumulative survival statistics for the entire series.

TABLE II. CUMULATIVE SURVIVAL
ALL RESECTIONS: 1933-1956

TIME PERIOD ENDING (YEARS)	DEATHS	DROPPED FROM STUDY	PATIENTS ENTERING	PATIENTS AT RISK	PERIOD MORTALITY	PERIOD SURVIVAL	CUMULATIVE SURVIVAL
1	148	0	341	341	43	57	57
2	46	0	193	193	23.8	76.2	43
3	37	5	147	144.5	25.6	74.4	32
4	9	4	105	103	8.7	91.3	30
5	6	8	92	88	6.8	93.2	28
6	5	10	78	73	6.8	93.2	26
7	3	4	63	61	4.9	95.1	25
8	6	5	56	53.5	11.2	88.8	22
9	3	6	45	42	7.2	92.8	20
10	3	10	36	31	9.7	90.3	18

For the sake of clarity results have been presented graphically in Figs. 4, 5, and 6. These show (1) the 5-year cumulative survival rate for the present series of resections compared with the original series, considering only patients who survived operation and therefore had the opportunity to survive (Fig. 4);

(2) the 10-year cumulative survival rate for the total series—all cases undergoing resection in the years 1933 to 1956 (Fig. 5); (3) the 5-year cumulative survival rates for "survey" cases compared with all resections for the years 1948 to 1956 (present series) (Fig. 6).

In the present series there was a 24 per cent 5-year cumulative survival rate for 127 pneumonectomies and a 33 per cent 5-year cumulative survival rate for 93 lobectomies. Detrimental to the survival rate of the pneumonectomy group is the higher operative mortality rate (10 per cent for pneumonectomy, 6.4 per cent for lobectomy) plus the higher incidence of "positive" lymph nodes (88) among the pneumonectomy patients (69 per cent) as opposed to 33 in the lobectomy group (36 per cent). Whether the lobectomy group would have fared better had they had pneumonectomy is speculative.

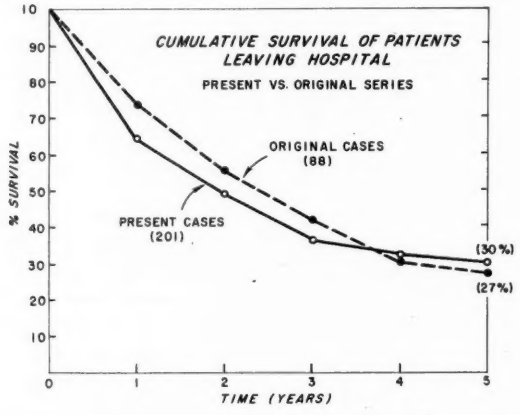

Fig. 4.

It is interesting to note that there has been no significant improvement in survival statistics in the present compared with the original series if the factor of operative mortality is excluded (see Fig. 4). It is reasonable to do this since many of the original series were operated upon during the developmental phase of surgery for pulmonary carcinoma. This should not be interpreted to imply that efforts to increase the extent of operation are futile.

Cumulative 10-year survival rates are shown in Fig. 5. There is an appreciable mortality in the 5- to 10-year period after operation. We cannot say with certainty how much of this mortality can be ascribed to age and intercurrent disease since perforce the majority of our death reports rely on clinical diagnosis and the tables are not comparable to ordinary life tables. However, many of the late deaths, probably more than half, were the result of recurrent or metastatic cancer. There is, as might be predicted, a flattening of the survival curve after 5 years and it is reasonable to conclude that 5-year survival is a significant measure of therapy in cancer of the lung.

The survival rate for "survey" cases is interesting in view of the optimistic hope now prevalent that earlier detection of cases will result in a greater percentage of survivals. The definition of "survey" cases is admittedly an approximate one. The 5-year cumulative survival rate for 38 "survey" cases operated upon in the years 1948 to 1956 was 42 per cent compared with 28 per cent for all resections carried out during the same period (Fig. 6).

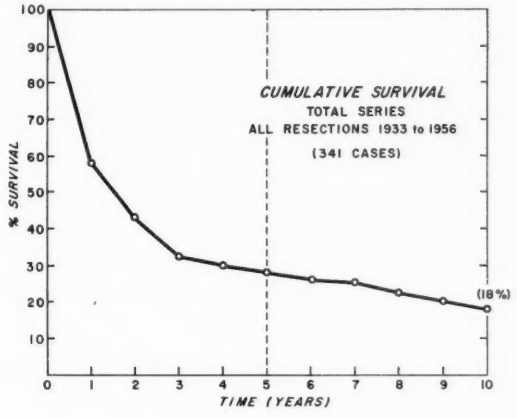

Fig. 5.

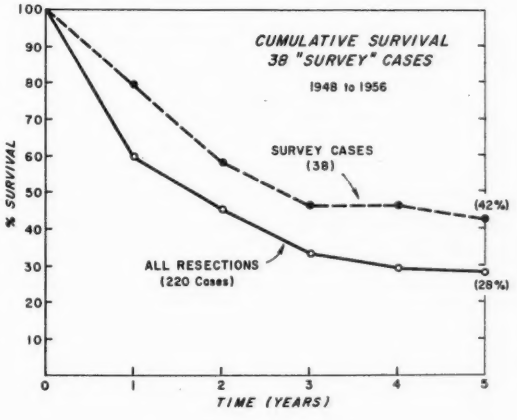

Fig. 6.

EFFECT OF POSITIVE NODES

One of the most difficult aspects of the cancer problem, whether it be of the lung or of any organ, is to measure accurately the importance of lymph node metastasis. There are an enormous number of variables. We have, therefore, been reluctant to dwell on this aspect of the problem in the present report.

Our material does not permit an accurate statement of number of nodes involved and whether or not these could be defined as intrapulmonary, regional, mediastinal, and other conventional designations. However, as a clinical measure, if we accept the notation of the pathologist who examined the specimen in a careful but "routine" way, we discover the following results. Of 56 patients undergoing pneumonectomy in the period 1948 to 1953 in whom at least one positive node was found, 9 were alive at 5 years (16 per cent); of 28 having pneumonectomies with "negative" nodes for the same period, produced 13 5-year survivals (46 per cent). Of 15 patients with positive nodes who had lobectomy in the same period, 3 (20 per cent) survived 5 years; of 43 patients with negative nodes and lobectomy 15 (35 per cent) survived 5 years.

SUMMARY

The experience with surgical resection of primary carcinoma of the lung at the Massachusetts General Hospital from 1950 to 1957 has been reviewed and added to a similar report from that hospital which covered the years 1930 to 1950. Complete follow-up data are available in all resected cases (341). These data show:

1. In the recent 7-year period, a greater percentage of patients with proved carcinoma of the lung have come to operation than in the earlier series (55 per cent compared with 43 per cent). The resectability rate has also increased (35 per cent of proved cases compared with 25 per cent).

2. The 5-year cumulative survival rate for all patients undergoing resection for cancer of the lung remains approximately 28 per cent.

3. The 10-year cumulative survival rate for such patients is 18 per cent.

REFERENCES

1. Churchill, E. D., Sweet, R. H., Soutter, L. and Scannell, J. G.: The Surgical Management of Carcinoma of the Lung, J. THORACIC SURG. **20:** 349-365, 1950.
2. Hill, A. B.: Principles of Medical Statistics, ed. 5, New York, 1950, Oxford University Press, chapt. 14, pp. 164-176.
3. Puffer, R. R.: Practical Statistics in Health and Medical Work, New York, 1950, McGraw-Hill, pp. 162-166.

(For Discussion, see page 324)

SHOULD WE INSIST ON "RADICAL PNEUMONECTOMY" AS A ROUTINE PROCEDURE IN THE TREATMENT OF CARCINOMA OF THE LUNG?

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AT A MEETING of the American Association for Thoracic Surgery in Denver in 1950, Allison, in discussing the paper of Churchill[1] and associates on cancer of the lung, proposed the term "radical pneumonectomy" to describe the removal of all possible mediastinal areolar tissue, containing the lymphatics, in continuity with the lung, thereby employing the principle previously utilized in operations for cancer in other parts of the body.

At about that time, Watson at the Memorial Hospital in New York became interested in the concept of radical pneumonectomy and subsequently published several papers on the subject.[2, 3] As described by Watson, the dissection starts at the apex of the mediastinum and all areolar tissue and lymph nodes are reflected downward as the trachea, esophagus, and other important structures, as well as the contralateral bronchus, are partially "skeletonized." The pericardium is resected with the lung and mediastinal tissues after the pulmonary vessels have been divided intrapericardially.

A few years ago the American Cancer Society gave wide circulation to the excellent motion picture made by Watson and his associates on the subject of radical pneumonectomy. This film implied that anyone doing less than a radical pneumonectomy for cancer of the lung might not be doing everything possible to cure his patients. We had been removing lymph nodes in the mediastinum immediately adjacent to the hilum, going over as far as the bifurcation of the trachea, but resected the more distant nodes only when there seemed to be involvement of those nodes and, therefore, found ourselves on the defensive regarding the surgical treatment of pulmonary cancer after viewing this film. When we asked our surgical friends around the country whether they performed radical pneumonectomies, they seemed hesitant to admit that they did not and if they did admit it, it seemed to us that they did so somewhat sheepishly.

The reason for feeling on the defensive concerning radical pneumonectomy is that it seems so reasonable, following the same concepts which led to the adoption of radical mastectomy for cancer of the breast and radical right

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colectomy for cancer of the cecum. If it is true that cancer spreads chiefly through lymphatic channels, it would seem that there could hardly be any disagreement on this subject. It is true that the lymphatic drainage tracts from the lung cross the mediastinum, making it difficult to remove all of the offending lymphatic channels even by radical pneumonectomy, but then the lymphatic channels from the breast often traverse the chest wall to enter the mediastinum, thus not being accessible in the ordinary radical mastectomy. The question naturally arises, will a super-radical pneumonectomy be proposed in which both pleural cavities are opened and the lymph node bearing tissue removed from both sides of the mediastinum, and the dissection perhaps carried up to and including the supraclavicular lymph node bearing areolar tissue? This super-radical operation has already been proposed for mammary cancer and has been carried out by some surgeons. It is obvious that it is up to the advocates of radical pneumonectomy for cancer of the lung to demonstrate that it produces better results than the standard type resection.

It should be pointed out that even the advocates of radical pneumonectomy do not use it routinely. Watson[2, 3] has limited its use to the patients who are considered good surgical risks and, indeed, only 55 per cent of his resections were classified as radical pneumonectomy.

We shall not belabor the point that radical pneumonectomy may carry a higher mortality than the standard procedures because it is difficult to compare mortality figures in various series done under various circumstances. However, it seems likely that, in general, the more radical the procedure for any condition, the higher the risk. When Allison discussed this point in 1950,[1] he mentioned that his mortality had been very high in his early experience with radical pneumonectomy, although in the year of 1949 he had been able to hold it down to less than 5 per cent. In his total series, however, the mortality was 23 per cent. Watson,[3] on the other hand, had a mortality of only 5.5 per cent in 74 radical pneumonectomies done from 1949 to 1951, whereas the mortality jumped to 25 per cent in 51 radical pneumonectomies done from 1952 to 1955. This seems high when one considers that only the good risk patients were subjected to radical pneumonectomy during that period. It also illustrates that mortality statistics may be unreliable in short series, since the difference between the 5.5 per cent and 25 per cent in consecutive 3-year periods is so striking. The total mortality for the 6-year period was 13.6 per cent. There can be no doubt that radical pneumonectomy has been carried out in the hands of a good many surgeons with a low mortality rate, at least for a period of time, but it seems probable that any thoracic surgeon who may be tempted to embark on such a program should give some consideration to the probability of increasing his mortality rate at least temporarily.

The decision as to whether radical pneumonectomy should be insisted upon, at least in good risk patients, must depend upon whether better results have been obtained than by the standard techniques. No one can deny that there is a certain attractiveness to the thesis that results might be improved by getting the last lymph node involved by lymphatic spread. It has now been

8 years since Allison proposed the term "radical pneumonectomy." As far as we know, Watson has published the largest series in which 5-year survival data are given. In his article published in 1956,[3] survival data were given on 116 patients operated upon during the years 1949 to 1951 (Table I). Seventy-four patients in that group were subjected to radical pneumonectomy with an operative mortality of 5.5 per cent and a 5-year survival of 27 per cent.

TABLE I. WATSON—5-YEAR SURVIVAL IN LUNG CANCER

RESECTION	NO.	% OPERATIVE MORTALITY	% 5-YEAR SURVIVAL
Radical pneumonectomy	74	5.5	27
Simple pneumonectomy	26	7.7	23
Lobectomy	16	18.7	25
Totals	116	7.7	26

Twenty-six patients in that same period had a standard pneumonectomy with a 5-year survival of 23 per cent, and 16 had a lobectomy with a 5-year survival of 25 per cent. In the total series, the 5-year survival was 26 per cent. Watson was encouraged to continue the use of radical pneumonectomy on the basis of the differential 5-year survival of 27 per cent for radical pneumonectomy and 23 per cent for standard pneumonectomy. When one considers, however, that it was the good risk patients who were subjected to radical pneumonectomy and that age is certainly one of the factors involved in the risk, it would not seem at all improbable that a higher percentage of the aged were subjected to simple pneumonectomy and that this might very well account for the lower 5-year survival rate in that group. It might also be suggested that the second 3-year period may not give as good a 5-year survival since the mortality for radical pneumonectomy in that period jumped from 5.5 to 25 per cent.

Gibbon and his associates[4, 5] have advocated an aggressive approach in resection for carcinoma of the lung. In their report covering 145 resections,[4] the vessels were divided intrapericardially in about 50 per cent. The report implies, however, that the radical operation was used for the extensive lesions rather than the early ones. They contended that lobectomy was justified only when the patient's condition would not permit a pneumonectomy.[5] The 5-year survival in their series of 145 resections was 21 per cent.

It seemed to us that it would be worth while to review our 5-year survival data since we have performed the standard type resection exclusively in our series. It is true that we have sometimes done more radical operations for carcinoma of the lung, ligating the vessels inside the pericardial sac and resecting large masses of nodes involved by tumor, but we have not "skeletonized" the trachea, the esophagus, and the great vessels in the mediastinum in good risk patients with early carcinoma. Our radical procedures have been confined to those patients who had tumors outside of the lung, making it necessary to carry out a more extensive resection in order to get around the palpable tumor. Thus, none of our patients can be said to have had a radical pneumonectomy in the sense that it was proposed by Allison.

The experience with carcinoma of the lung at the Hospital of the University of Pennsylvania from Jan. 1, 1939. to Jan. 1, 1958, was with 764 patients. Fifty-one per cent were explored and 33 per cent were subjected to resection. The operative mortality was 8.6 per cent. Not all of these patients had histologic proof of carcinoma of the lung. Some, who were not subjected to operation, had a clinical diagnosis only and many of them were never seen by our surgical group. However, it was the clinical impression that these patients did have carcinoma of the lung and we present these data regarding the entire series in an effort to show the true ravages of the disease rather than confining ourselves only to those cases which were seen by us personally and histologically proved. Because of this, the figure of 33 per cent of the patients of the entire series being subjected to resection may seem a bit low as compared with other series, but 65 per cent of those explored were subjected to resection and this is in accord with other reported series. In this report we have confined our attentions to the patients seen more than 5 years ago. In the period from Jan. 1, 1939, to April 1, 1953, a total of 344 patients who clinically were judged to have cancer of the lung were seen at the Hospital of the University of Pennsylvania (Table II). Of these, 192 (56 per cent) were explored, and 116 (34 per cent) were subjected to pulmonary resection. There were 9 deaths, giving an operative mortality of 7.7 per cent. Of this group of 116 patients,

TABLE II. CARCINOMA OF LUNG JANUARY 1, 1939—APRIL 1, 1953

	NO. OF PATIENTS	%	5-YEAR SURVIVAL	
			NO.	%
Clinical diagnosis	344			
Explored	192	56.		
Resected	116	34.	31	26.7
Operative mortality	9	7.7		
Pneumonectomy (standard)	99	7.	26	26.4
Less than pneumonectomy	17	13.	6	35.

31 lived for 5 years or more, giving a 5-year survival rate of 26.7 per cent of those subjected to resection or 9 per cent of all patients judged clinically to have carcinoma of the lung. In the group subjected to resection 99 had pneumonectomy with 26 per cent 5-year survival, and 17 had less than a pneumonectomy with 35 per cent 5-year survival. It will be seen that our 5-year survival rate of 26.7 per cent is comparable to that of Watson's 27 per cent and is essentially the same as Gibbon's 21 per cent if one takes into consideration the difference in operative mortality. Using the published 5-year data from the six clinics[1, 2, 4, 6, 7] shown in Table III, it would appear that the 5-year survival rate tends to show an inverse relationship to the operative mortality.

It is admittedly rather early to be judging the results of radical pneumonectomy since the number of patients operated upon by that technique more than 5 years ago is still comparatively small. At the present time, however, and until further data are available, it cannot be said that radical pneumonectomy is a superior operation to the standard type resection from the standpoint of 5-year survival.

TABLE III. 5-YEAR SURVIVAL—CARCINOMA OF LUNG

SENIOR AUTHOR AND YEAR	NUMBER RESECTED	% OPERATIVE MORTALITY	% RESECTED PATIENT 5-YEAR SURVIVAL	% TOTAL PATIENT 5-YEAR SURVIVAL
Churchill 1950	69	19.9	14.5	
Overholt 1956	234	15.	22.	7.6
Ochsner 1956		19.2	15.	5.5
Watson 1956				
Radical pneumonectomy	74	5.5	27.	
Less than radical pneumonectomy	42	11.	23.8	
Gibbon 1956	145	23.	21.	8.4
Johnson 1958	116	7.7	26.7	9.

It has only been within the last few years that we have had a clue which may help to explain why radical pneumonectomy, at least to date, has not been shown to be superior to the standard type resection for cancer of the lung. We believe the explanation may lie in recent evidence regarding the spread of pulmonary cancer through the blood stream as well as through the lymphatic channels. Dr. Fred Collier, until recently one of our pathologists, became interested in this problem.[8, 9] By means of special stains he studied the surgical specimen for evidence of blood vessel invasion. He was interested in knowing whether the presence or absence of blood vessel invasion in the surgical specimen could be correlated with the survival. In this study he found demonstrable blood vessel invasion in 71 per cent of the surgical specimens. Further studies[10] indicated that blood vessel invasion was of far greater prognostic significance than the presence or absence of lymph node invasion. For many years surgeons[11] have been aware that there is considerable difference in the cellular type of tumors and that some tumors spread rapidly and others spread slowly. It seems to us that Collier and others who have studied this problem may have disclosed a crucial factor in the widespread dissemination of these tumors. As has previously been reported,[10] it was found that undifferentiated carcinomas all showed blood vessel invasion, whereas only 63 per cent of the epidermoid carcinomas, 45 per cent of the bronchiolar carcinomas, and 12 per cent of the carcinoid adenomas showed blood vessel invasion. The presence or absence of blood vessel invasion has a profound influence upon 5-year survival. In our present series (Table IV), of 71 patients with blood vessel invasion, only 4 (6 per cent) survived 5 years, whereas of 36 patients without blood vessel invasion, 27 (75 per cent) survived 5 years. This is of much greater significance than the presence or absence of lymph node invasion. For example, of 53 patients with lymph node invasion, only 8 (15 per cent) survived 5 years, whereas of 54 patients without lymph node invasion, 23 (43 per cent) survived 5 years. On the other hand, if the patient had blood vessel invasion, the presence or absence of lymph node invasion had no significance. In the absence of blood vessel invasion, the presence of lymph node invasion was of some significance since of 10 such patients, only 6 (60 per cent) survived 5 years. The best results were in the group of 26 patients with neither blood vessel nor lymph node invasion in which 21 (81 per cent) survived 5 years.

TABLE IV. CARCINOMA OF LUNG—5-YEAR SURVIVAL OF 107 PATIENTS SURVIVING RESECTION

	BLOOD VESSEL INVASION	LYMPH NODE INVASION	RESECTED	ALIVE 5 YEARS	PER CENT
1.	+		71	4	6
2.	–		36	27	75
3.		+	53	8	15
4.		–	54	23	43
5.	+	–	28	2	7
6.	+	+	24	2	8
7.	–	+	10	6	60
8.	–	–	26	21	81

If these studies of Collier and his associates are confirmed by other groups, perhaps we should revise our thinking regarding carcinoma of the lung. Formerly, spread through lymphatic channels appeared to be the most reliable prognostic criteria and it seemed reasonable to extend the scope of the operation in the hope of getting around all lymphatic spread. Now it seems likely that the spread through the blood stream is of greater significance but that extension of the scope of the resection cannot possibly influence this factor. As far as we know, these observations on the relationship of blood vessel invasion to 5-year survival have not yet been confirmed in other clinics. It is of some interest to note, however, that whereas our pathologists found 71 per cent of the surgical specimens to show blood vessel invasion, Moore and his associates,[12] who have used a technique for demonstrating cancer cells in the blood stream, have found cancer cells in approximately 75 per cent of the patients with cancer of the lung.

At the present time there are no data available to indicate when blood vessel invasion occurs in a cancer of the lung. We can only say that we have seen it present in small tumors and absent in large ones. Our data would suggest that if it is present in a small tumor, even a radical pneumonectomy is not likely to save the patient from widespread dissemination of the lesion. On the other hand, the fact that it may be absent in a large tumor has spurred us on to make an increased effort to get around such a tumor when it is technically possible to do so. It would appear obvious that if there is not blood vessel invasion and if the tumor has spread through the lymphatics, a radical pneumonectomy might offer a better chance of getting around "that last lymph node" which may be involved. Our fear has been that the occasional gain made on this basis might be more than offset by an increase in the mortality.

SUMMARY

1. In an experience with 764 patients with carcinoma of the lung at the Hospital of the University of Pennsylvania, 344 were seen more than 5 years ago. Of this group, 192 (56 per cent) were explored and 116 (34 per cent) were subjected to pulmonary resection. The operative mortality was 7.7 per cent. Thirty-one or 26.7 per cent of those subjected to resection, or 9 per cent of the total number of patients, survived 5 years or more. "Radical" pneumonectomy was not employed in this series.

2. The 5-year survival so far reported following "radical" pneumonectomy is not significantly higher than we have obtained with the standard type resection.

3. It is suggested that until it can be shown that "radical" pneumonectomy produces superior results, the thoracic surgeon is not obligated to perform the radical operation. However, we do not wish to discourage the resection of all grossly involved tumor tissue and feel that the surgeon should make a real effort to get around the locally invasive tumor whenever possible.

4. Studies to date indicate that blood vessel invasion as found in the surgical specimen is of greater prognostic significance than lymph node invasion. Whereas there was only a 6 per cent 5-year survival in those patients showing blood vessel invasion in their tumor, there was a 75 per cent 5-year survival when blood vessel invasion was not present. This may in part account for the fact that radical pneumonectomy to date has not produced the great improvement in the 5-year survival rate which was anticipated.

We wish to express our appreciation to Dr. Samuel Askowitz and Miss Mary Jane English for their assistance in compiling the statistical data.

REFERENCES

1. Churchill, E. D., Sweet, R. H., Soutter, L., Scannell, J. G.: Surgical Management of Carcinoma of the Lung, J. THORACIC SURG. **20**: 349-365, 1950.
2. Watson, W. L.: Carcinoma of the Lung With Five Year Survivals: A Study of 3,000 Cases, J. Internat. Coll. Surgeons **26**: 750-754, 1956.
3. Watson, W. L.: Radical Surgery for Lung Cancer. Evaluation of the Operation of Radical Pneumonectomy and Five Year End Results, Cancer **9**: 1167-1172, 1956.
4. Gibbon, J. H., Templeton, J. Y., and Nealon, T. F.: Factors Which Influence the Long Term Survival of Patients With Cancer of the Lung, Ann. Surg. **145**: 637-641, 1957.
5. Gibbon, J. H., Jr., and Nealon, T. F., Jr.: Carcinoma of the Lung and Tumors of the Thorax *in* Surgery: Principles and Practice, Philadelphia, 1957, J. B. Lippincott Co., chap. 44.
6. Overholt, R. H., and Bougas, J. A.: Fifty-one Cases of Lung Cancer With Five Year Survival, J. A. M. A. **161**: 961-963, 1956.
7. Ochsner, A.: Cancer of the Lung, Postgrad. Med. **19**: 584-592, 1956.
8. Collier, F. C. D., Kyle, R. H., Enterline, H. T., Tristan, T., and Greening, R.: Pulmonary Carcinoma: Correlation Between Morphology and Survival, Am. J. Path. **33**: 582, 1957 (Abst.).
9. Collier, F. C. D., Kyle, R. H., Enterline, H. T., Tristan, T., and Greening, R.: Presentation Before the Meeting of the American Trudeau Society, May 6-8, 1957, Kansas City, Mo.
10. Collier, F. C. D., Blakemore, W. S., Kyle, R. H., Enterline, H. T., Kirby, C. K., and Johnson, J.: Carcinoma of the Lung: Factors Which Influence Five Year Survival With Special Reference to Blood Vessel Invasion, Ann. Surg. **146**: 417-423, 1957.
11. Kirklin, J. W., McDonald, J. R., Clagett, O. T., Moersch, H. J., and Gage, R. P.: Bronchogenic Carcinoma: Cell Type and Other Factors Relating to Prognosis, Surg., Gynec. & Obst. **100**: 429-438, 1955.
12. Moore, G. E., Sandberg, A., and Schubarg, J. R.: Clinical and Experimental Observations of the Occurrence and Fate of Tumor Cells in the Blood Stream, Ann. Surg. **146**: 580-587, 1957.

(For Discussion, see page 324)

RESULTS IN THE TREATMENT OF BRONCHOGENIC CARCINOMA

An Analysis of 1,008 Cases

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One thousand and eight patients with primary carcinoma of the lung were seen by the senior author and his associates at the Barnes Hospital between Jan. 1, 1948, and Dec. 31, 1955. Appropriate data were obtained from the total case record of each of the 1,008 patients for this analysis. The diagnosis was verified in 100 per cent of the cases by microscopic section, the material being obtained by bronchoscopic biopsy, sputum cytology, biopsy of metastatic implants, needle biopsy, frozen section biopsy at the time of thoracotomy, and examination of the resected specimens.

The date of follow-up was July 1, 1957. The minimum follow-up period of this group was therefore 1½ years and the maximum 9½ years. No autopsy cases not seen and treated are included. A larger experience dating from the first pneumonectomy for carcinoma by Graham in 1933 to 1948 is not included. This earlier group for various reasons does not lend itself to accurate statistical analysis.

TABLE I. DISTRIBUTION OF LUNG CANCER BY AGE

AGE	NUMBER	PER CENT
20-29	2	
30-39	22	2
40-49	164	17
50-59	375	37
60-69	361	36
70-79	80	8
80-89	4	
Totals	1,008	100

SEX AND AGE

The ratio of men to women did not differ significantly from other reported series.[2, 4, 7] There were approximately 8 times as many men (88.4 per

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cent) as women (11.6 per cent). Age at the time of treatment ranged from 24 years to 85 years. The highest incidence was encountered in the fifth and sixth decades (Table I).

PATHOLOGY

While minor differences exist among surgical pathologists in the description of various types of lung cancer, most agree on a simple classification. The Surgical Pathology Division at the Barnes Hospital recognizes the following classification:

A. Epidermoid carcinoma
B. Undifferentiated carcinoma
C. Adenocarcinoma
D. Bronchiolar (alveolar cell) carcinoma
E. Small (oat) cell carcinoma.

All cases of bronchial adenomata have been excluded from this series.

CELL TYPE

The histologic nature of the tumors seen in this series proved very similar from the point of view of incidence to those reported from other centers.[3] The majority proved to be epidermoid (57 per cent). Undifferentiated carcinoma made up 28.5 per cent of the group, and adenocarcinoma 10 per cent (Table II).

TABLE II. HISTOLOGIC DIAGNOSIS

TYPE	NUMBER	PER CENT
Epidermoid	572	57.0
Undifferentiated	287	28.5
Adenocarcinoma	107	10.5
Bronchiolar	13	1.0
Small (oat) cell	15	1.5
Mixed epidermoid and adenocarcinoma	14	1.5
Totals	1,008	100.0

SITE

The right lung was the site of the cancer in 580 patients, and the left in 428. The tumor arose in the upper lobes in 485 (48 per cent) patients. The right upper lobe was the most frequent site (25 per cent). The distribution is shown in Table III.

TABLE III. SITE OF THE LUNG CANCER

BRONCHUS	NUMBER	PER CENT
Right main	85	8.5
Right upper	252	25.0
Right middle	39	4.0
Intermedius	49	5.0
Right lower	155	15.0
Left main	86	8.5
Left upper	233	23.0
Left lower	109	11.0
Totals	1,008	100.0

SELECTION OF CASES

In the 1,008 patients, 390 or 38.6 per cent were not considered suitable for exploration (Fig. 1). In each of these cases the diagnosis was verified by appropriate biopsy material. The vast majority were inoperable because of generalized spread of tumor, including diffuse cerebral or bony metastases, bilateral pulmonary involvement, carinal, tracheal or chest wall extension. The number considered inoperable because of pulmonary insufficiency was small because of our policy of advising exploration on all so-called borderline cases as judged by function studies and clinical evaluation. Likewise very few patients were turned down because of co-existent cardiac or renal disease. In our experience, intractable heart failure is the only cardiorenal condition in which surgery almost invariably leads to serious difficulty. Age was not a deterrent. In only 2 patients, both in the 80's, was surgery not advised because of infirmity.

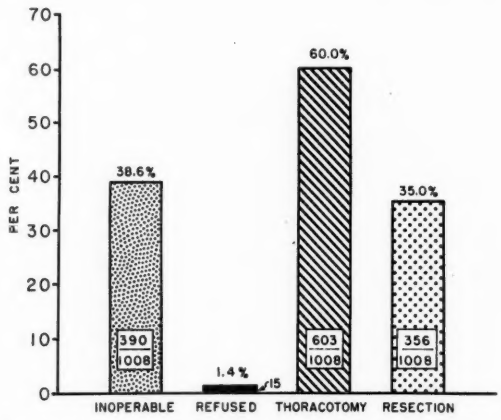

Fig. 1.—Management of 1,008 cases of bronchogenic carcinoma.

Our policy concerning exploration in the special instances of pulmonary cancer has always favored the side of surgical intervention whenever possible. This applies to cases of phrenic or recurrent laryngeal nerve involvement, chest wall involvement without extensive bony destruction, and to certain cases with positive scalene lymph node biopsy or positive pleural fluid. Surgery in many of these instances is obviously palliative. The results of such procedures defy statistical analysis, since the patient may not live a day longer than the natural course of the disease would allow, but if the patient is done no harm by operation and the quality of his remaining existence is improved, this certainly is as great a surgical dividend as prolongation of life. Surgeons must remember that life for the cancer patient is a matter of quality not quantity. For this reason surgeons treating pulmonary cancer must not let themselves be hampered by hard and fast rules of operability and inoperability. In this regard, experience and judgement count far more than the rule book. For

example, those who contend that a positive scalene node biopsy is a contraindication to surgery will be doing a number of patients a grave injustice in terms of ultimate comfort.

RESECTABILITY RATES

In addition to the 38.6 per cent considered clinically inoperable, 15 patients (1.4 per cent) refused surgery (Fig. 1). The remaining 603 patients (60 per cent) had exploratory thoracotomy, and resection was done in 356 of them. Thus, the resection rate for the entire 1,008 cases was 35 per cent. In the other 247 explorations, the tumor was found to be nonresectable, the majority because of invasion of hilar blood vessels.

Pneumonectomy was the preferred operative procedure for all cell types except bronchiolar carcinoma, for which lobectomy was favored. Of the 356 resectable cases, 280 or 79.3 per cent, had pneumonectomies, 74 (20.2 per cent) had lobectomies, and 2 (0.5 per cent) had segmental resection (Fig. 2). Our figures show that in spite of a preference for pneumonectomy, the percentage of lobectomy cases has been steadily increasing. Whereas in 1948 lobectomy comprised only 3.3 per cent of the total resections, in 1955 the percentage had

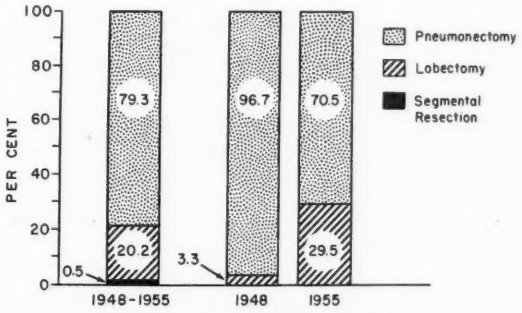

Fig. 2.—Type of resection.

increased to 29.5 per cent. We attribute this rise to the extended use of lobectomy in palliation and in older patients with borderline respiratory reserve, plus its use in selected cases of small peripheral solitary tumors.[8] This latter group has not been large in our series. In the 2 cases of segmental resection, 1 was a mistaken frozen section diagnosis in which re-operation was not advised, and the other was a palliative resection for a peripheral tumor with involvement of the chest wall and pain in a poor risk patient.

OPERATIVE MORTALITY

There were 69 deaths (11.0 per cent) within the first 2 months following surgical exploration (Table IV). Approximately one half of the deaths were due to cardiovascular complications. The routine use of prophylactic preoperative digitalization during the past few years has significantly reduced the number of cardiovascular disturbances which endanger the life of the older age

patient. In the group in which the tumors were found nonresectable at operation, the mortality was 8.8 per cent. Deaths in cases of pneumonectomy totaled 13.0 per cent, and in the lobectomy cases 12.0 per cent.

TABLE IV. OPERATIVE MORTALITY

OPERATION	NUMBER OF CASES	NUMBER OF DEATHS	PER CENT
Exploration	247	22	8.8
Pneumonectomy	280	37	13.0
Lobectomy	74	9	12.0
Segmental resection	2	1	50.0
Totals	603	69	11.0

SURVIVAL RATES

In analyzing these results, those patients who were never heard from, once they left the hospital, are not included (4.4 per cent). In cases where the patient was heard from or seen months or years later and then no further follow-up obtained, it was assumed that the day the patient was last seen or heard from was the expiration date, and that death was due to the cancer.

Of 1,008 cases of primary carcinoma of the lung in this series, 482 patients were treated between January, 1948, and July, 1952. Of these, follow-up was obtained in 461 (95.6 per cent), which represents the total number of cases

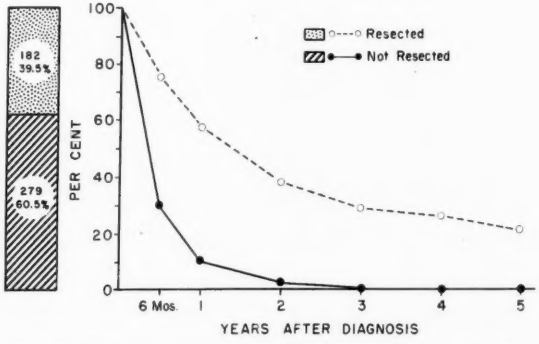

Fig. 3.—Survival—461 cases. January, 1948—July, 1952.

available for survival analysis 5 to 9½ years after diagnosis (Fig. 3). Resections were performed in 182 of these 461 cases, a resectability rate of 39.5 per cent. The 5-year survivors in these 182 cases numbered 40 patients, a survival rate of 22 per cent. Seventy patients (38.4 per cent) lived more than 2 years following operation. The critical period of survival in this series was the first 30 months after resection. Of the patients who survived at least 30 months, 75 per cent lived on to become 5-year survivors.

There were no 5-year survivors in the remaining 279 inoperable or nonresectable patients, even though almost all of them were treated with x-ray therapy or chemotherapy or both. Seventy per cent of these patients were dead within 6 months, and only 4 patients, or 1.4 per cent, were alive after

2 years, which again emphasizes the necessity of recommending surgery if at all possible in the treatment of this disease. Thus, 40 persons out of the original group lived 5 or more years, an over-all survival rate of 8.7 per cent.

An analysis of the resected specimens on these 40 long-term survivors showed, as would be expected, that epidermoid carcinoma was the predominant cell type, occurring in 31 of the 40 cases (Fig. 4). There were 5 cases of undifferentiated carcinoma, representing 12.5 per cent of the total number. This

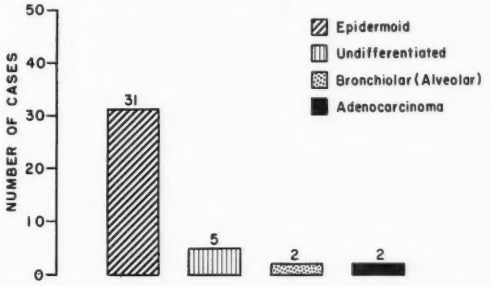

Fig. 4.—Cell type—40 cases with 5-year survival.

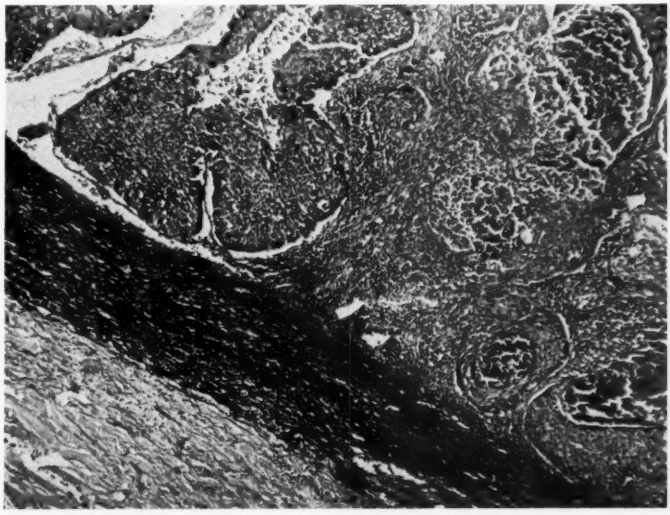

Fig. 5.—Photomicrograph showing invasion of the main pulmonary artery by epidermoid carcinoma. The vessel wall with stained elastic fibers is at the lower left. This patient is alive and well more than 5 years following resection (Verhoeff-van Gieson, ×125).

gratifying finding increases our optimism about this type of cancer of the lung, and certainly leads us to take exception with those who believe that patients with known undifferentiated carcinoma should not be treated surgically. There were 2 cases each of bronchiolar cell tumor and adenocarcinoma.

The 40 specimens were further analyzed for frequency of lymph node and blood vessel invasion (Table V). A totally unexpected finding was that 18 of

the 40 cases, or 45 per cent, had involvement of lymph nodes by tumor. The number of involved nodes in these 18 cases ranged from 1 to 7, with the average being 2½ nodes per case. Three of the 40 cases, 8 per cent, had obvious blood vessel invasion on routine hematoxylin and eosin stains, and 2 of these 3 cases also had lymph node involvement. This high proportion of survivors with tumor extension to lymph nodes is unusual, since most survival statistics show the great preponderance of patients living 5 years have negative nodes.[2, 5] It has recently been shown that blood vessel invasion may be a more important factor in determining prognosis than lymph node invasion[1] (Fig. 5).

TABLE V. EXTENSION OF TUMOR—40 CASES WITH 5-YEAR SURVIVAL

Lymph node involvement	18 cases—45%
Range per case	1 to 7 nodes
Average per case	2.5 nodes
Blood vessel invasion	3 cases— 8%

From July, 1952, to December, 1955, a diagnosis of pulmonary cancer has been made in 522 cases, and 496 (95 per cent) have been followed from 1½ to 5 years (Fig. 6). The ultimate long-term survival figures are of course not complete, but it is interesting to compare trends in this group of cases with the

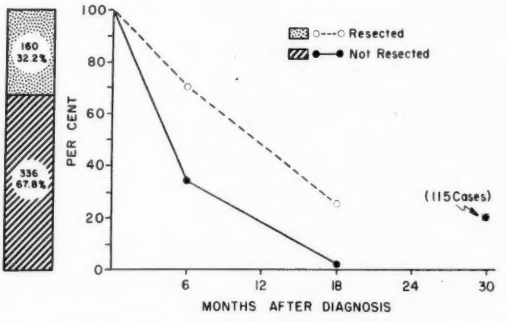

Fig. 6.—Survival—496 cases. July, 1952—December, 1955.

5 to 9½ year group. The number of patients we are seeing with cancer of the lung is increasing each year. In the earlier period—spanning 4½ years—461 cases were diagnosed; whereas in the more recent period covering only 3½ years, 496 cases were seen. There is no indication that lesions are being seen at an earlier stage, for the resection rate is actually lower in the 1952-1955 group, 32.3 per cent, compared with 39.5 per cent for 1948-1952. There is likewise no indication that any significant refinement or extension of surgical techniques has been achieved, since comparison of percentage survival of resected cases in the 2 groups is essentially the same. Of the 160 patients resected from July, 1952, on, 115 have been followed a minimum of 30 months and, of these, 20 per cent are alive. In the 1948-1952 group the comparable figure for 30-month survival was 25 per cent.

DISCUSSION

This study once again re-affirms the discouraging uniformity of results that are being obtained throughout the country in the treatment of pulmonary cancer. Table VI shows clearly the fundamental problem: Only one third of all the patients who develop carcinoma of the lung are surgically resectable and thus eligible for long-term survival. At Barnes Hospital there has been no change in the percentage of resectable cases over the past 9½ years. Improvements have come in many areas in recent times. The mortality rates for surgery are quite low, the poor risk patients are being operated upon more successfully, and so forth. But until a larger number of the total case group can be seen at a time when the lesion can be removed, it is unlikely that the 5-year survivor group will ever get over 10 per cent. We do not feel on the other hand that there is cause for too much pessimism or that talk of fate or biologic predeterminism should cloud the issue.[6] The high incidence of 5-year survival we obtained in cases of undifferentiated carcinoma, and in cases with gross lymph node and blood vessel invasion, has reinforced our feeling that the most important step in curing cancer is still the surgical removal of all tumor-containing tissue, and that all patients should be considered for resection if there is reasonable belief that this can be done. It is certainly true that there are some cases of pulmonary cancer where growth is so rapid and invasive that the patient is going to die no matter when the diagnosis is made. It is also true that in other cases growth is so slow that the diagnosis can be missed by several physicians and still a good result will ensue. But the vast majority of cancers of the lung we have seen progress in an orderly fashion, and without undue rapidity—from curable to the incurable stage. This being the case it should be possible with all the diagnostic aids and knowledge at hand to discover them in the phase of growth when resection can be done on the majority.

TABLE VI

AUTHOR	NO. CASES	PER CENT EXPLORED	PER CENT RESECTED	PER CENT 5-YEAR SURVIVALS
Burford	1,008	60	35	9
Gibbon	532	71	39	9
Kirklin	767	48	24	8
Ochsner	1,170	52	33	8
Overholt	733	62	37	8

SUMMARY

1. One thousand and eight histologically confirmed cases of bronchogenic carcinoma have been seen between Jan. 1, 1948, and Dec. 31, 1955.

2. The distribution as to sex, age, cell type, and site of origin did not differ significantly from other reported series.

3. Surgical exploration was carried out in 60 per cent of cases. Resection was possible in 35 per cent. Pneumonectomy comprised 80 per cent of the resections, lobectomy 20 per cent. The percentage of lobectomy cases increased from 3 per cent in 1948 to 30 per cent in 1955.

4. The over-all operative mortality was 11 per cent.

5. The 5-year survival rate for all patients followed an adequate length of time was 9 per cent. In the select group in whom surgical resection was possible, the 5-year survival rate was 22 per cent.

6. Study of the resected specimens from the 5-year survivors revealed epidermoid carcinoma to be the most prevalent cell type (77.5 per cent), but a surprisingly high number of undifferentiated tumors (12.5 per cent) were present. Almost half (45 per cent) of the specimens showed lymph node involvement, and 8 per cent had blood vessel invasion.

7. Surgical resection could be done in only 1 of each 3 diagnosed cases. This ratio remained unchanged throughout the 8-year span of this series. Significant increase in 5-year survivals will be possible only through an increase in the percentage of resections performed.

REFERENCES

1. Collier, F. C., Blakemore, W. S., Kyle, R. H., Enterline, H. T., Kirby, C. K., and Johnson, J.: Carcinoma of the Lung: Factors Which Influence Five Year Survival With Special Reference to Blood Vessel Invasion, Ann. Surg. **146:** 417, 1957.
2. Gibbon, J. H., Allbritten, F. F., Templeton, J. Y., and Nealon, T. F.: Cancer of the Lung—An Analysis of 532 Consecutive Cases, Ann. Surg. **138:** 489, 1953.
3. Kirklin, J. W., McDonald, J. R., Clagett, O. T., Moersch, H. J., and Gage, R. P.: Bronchogenic Carcinoma: Cell Type and Other Factors Relating to Prognosis, Surg., Gynec. & Obst. **100:** 429, 1955.
4. Ochsner, A., Ray, C. J., and Acree, P. W.: Cancer of the Lung, Am. Rev. Tuberc. **70:** 763, 1954.
5. Overholt, R. H., and Bougas, J. A.: Fifty-one Cases of Lung Cancer with Five Year Survival, J. A. M. A. **161:** 961, 1956.
6. Paulson, D. L.: Survival Rates Following Resection for Bronchogenic Carcinoma, Ann. Surg. **146:** 997, 1957.
7. Rienhoff, W. F., Jr.: A Clinical Analysis and Follow-up Study of Five Hundred and Two Cases of Carcinoma of the Lung, Dis. Chest **17:** 33, 1950.
8. Robinson, J. L., Jones, J. C., and Meyer, B. W.: Indications for Lobectomy in the Treatment of Carcinoma of the Lung, J. THORACIC SURG. **32:** 500, 1956.

DISCUSSION

(PAPERS BY CHURCHILL [PAGE 301], JOHNSON [PAGE 309], AND BURFORD [PAGE 316] AND THEIR ASSOCIATES)

DR. RICHARD H. MEADE, Grand Rapids, Mich.—We have heard three excellent papers on a very important subject. I was fortunate in being present at the meeting in 1933 when Dr. Graham reported his case. It is rather interesting that he was not on the program and, after some of the papers had been read, he rose and said, "Gentlemen, I think you will be interested in something I did last week." He then told us about this case. I think we should remember that the longest surviving patient with carcinoma of the lung, operated upon by Dr. Graham 25 years ago, had the most conservative type of pneumonectomy possible. There was no attempt to remove adjacent glands, and some lung tissue was left around the stump.

It occurs to me that the best way to study this problem is not shown in the series of cases presented today but rather from autopsies of patients who have had successful resections, to see what happened to them. Through the co-operation of Sir Clement Price Thomas in London, we have been able to study 73 cases; 50 of these were simple pneumonectomies and 23 were lobectomies. The average length of life at autopsy was 20 months; this interval varied from 3 months up to 96 months, the longest being 8 years before death.

There was no recurrence of cancer in 32 per cent at the time of autopsy. There was recurrence in ipsilateral glands in 28 per cent and in contralateral glands in 4 per cent. There was no recurrence in the pulmonary vessel stump in any case, and three recurrences in the pericardium.

In the 23 lobectomies there was no recurrence in 26 per cent; there was recurrence in the ipsilateral glands in 21 per cent, and no recurrence in the pericardium.

It is my hope that during the course of the next year I may have the co-operation of this Association so that a representative series may be studied. I believe this is a very interesting way to approach the problem, to see the end results of pulmonary resection.

DR. GABRIEL P. SELEY, New York, N. Y.—I would like to discuss the paper of Dr. Johnson. For a number of years at the Mt. Sinai Hospital in New York, Dr. Otani has studied all specimens removed for cancer of the lung, with particular reference to blood vessel invasion. He has found a great number of cases of blood vessel invasion, but particularly adenocarcinomas. As a matter of fact, in every case of carcinoma of the lung with the histologic classification of adenocarcinoma, there has been definite blood vessel invasion. I also wish to bring to your attention the fact that in prognosis of cancer of the lung, the histology is extremely important, and you will notice in Dr. Burford's figures there was an extremely high mortality rate and very poor survival rate in the adenocarcinoma group. I think one should take these things into consideration by doing very careful studies including neurological examinations, electroencephalograms, and bone surveys when one suspects an adenocarcinoma of the lung. In addition to this, as to the specific technique at the time of operation, one should respect these tumors, manipulate them as little as possible and, as a matter of fact, one should make every effort in all cases of carcinoma of the lung, but particularly adenocarcinoma, to first isolate the veins and ligate them before any manipulation of the tumor itself.

I would like to ask Dr. Johnson whether, in his experience with the blood vessel invasion cases, the adenocarcinomas have had such a high invasion rate as we have found at Mt. Sinai Hospital.

DR. ALFRED HURWITZ, Brooklyn, N. Y.—I rise solely to take mild issue with Dr. Burford on one aspect of his discussion when he implied that other than the overt, surgeon-induced complications, surgery for carcinoma of the lung can do no harm. Unfortunately, I have had a few patients whose postoperative course has been characterized by the rapid development of disseminated metastases and death. Some of these patients have been followed by their physicians for 2 to 4 years and have had minimal symptoms and little if any change in the size of their lesions. Rigler has described some interesting cases of bronchogenic carcinoma where there has been little or no change in size on x-ray for as long as 5 and 6 years. Paulson discussed our present dilemma and pointed out that it would be a greet boon to mankind if we could develop some biochemical test which would help us to decide which patients would react adversely to surgery and which ones would be benefited by excisional surgery. I am convinced that at times we may destroy the patient's biologic resistance or state of predeterminism which he may have amassed over a long period.

I also noticed one omission in all three papers. None of the speakers discussed their criteria for defining operative mortality. I hope they will define what they mean by operative mortality.

DR. IRVING A. ~~SHORT~~ Sarot, New York, N. Y.—In 1934, Dr. Rabin and Dr. Neuhof called attention to the fact that carcinomata of the lung which appeared as circumscribed shadows on the roentgenogram were much more likely to be operable than those which did not produce a discrete circumscribed shadow. My study, in 1941, confirmed this observation.

The question has arisen as to what influence the microscopic cell type has on operability. This was studied in a series of 426 consecutive cases by Drs. Ragin, Schein, Rubinstein, and myself. There were 117 circumscribed tumors and 309 non-circumscribed ones.

Although the cell type does influence operability, the circumscribed tumors are much more apt to be operable in each cell type. Thus, in the mature squamous carcinoma, which is the most favorable, the percentage of operability of the circumscribed tumors was twice as great as the non-circumscribed ones. The difference was even more striking in the tumors of the less favorable cell types; namely, the immature squamous, adeno-, anaplastic, and small cell carcinomas. In these types, the ratio was 6 to 1 in favor of the circumscribed tumors.

It must be concluded that the circumscription of the growth is a more accurate index of operability than the cell type.

DR. WILLIAM G. CAHAN, New York, N. Y.—Dr. Graham showed that it was possible to cure cancer of the lung by surgery. Long-term survivals have been reported, not only after simple pneumonectomy but after lobectomy and wedge resection as well. This suggests that there must be various elements in survival apart from the magnitude of the operation. Nevertheless, in 1949, I wondered if there would be any improvement in chances of cure if the mediastinal lymph nodes were added to pulmonary resection as part of radical lobectomy or radical pneumonectomy.

At the 1951 meeting of this Association, as a group, we presented the rationale and step-by-step technique of our version of a radical pneumonectomy. This block dissection was based upon principles of cancer surgery elsewhere within the body. It seemed particularly applicable to lung cancer, for pathologists had been unanimous in describing mediastinal lymph nodes as being the most frequent sites of metastasis, regardless of size, position, and histologic type of the primary tumor.

At Memorial Center, we have performed 150 radical pneumonectomies and over half of these were done by residents as their first pneumonectomy. It is too soon to evaluate the procedure thoroughly, particularly in the light of variables that surround the behavior of lung cancer. After all, radical mastectomy is still controversial after 60 years! But we have been puzzled by certain attitudes about the mediastinal nodes at the time of thoracotomy. Why, for example, do some surgeons still rely upon palpation as the sole method of detecting the presence or absence of metastases? Why do others select a mediastinal lymph node for frozen section and, if this proves to be positive, assume all is hopeless; or if it proves to be negative, consider further lymphatic dissection needless? They would certainly not apply these principles to an abdominoperineal resection or radical mastectomy, for example.

Now it is said that venous invasion may be more significant in prognosis than lymph node involvement, but this may be said also to be true of other sites of cancer, and yet no lesser procedure is performed. In any event, unless grossly evident, venous invasion can only be found in the postoperative specimen and, as such, it cannot in any way influence the type of procedure performed; whereas lymph nodes may be excised or left behind. In addition, we have seen survivors of 5 or more years who have shown blood vessel invasion in the excised specimens, but we know of no such survivor where proved positive mediastinal lymph nodes were allowed to remain. Remember, positive lymph nodes left in situ are not dormant; they can serve not only as sources of invasion, but are capable of giving rise to second generation—or daughter—metastases as well.

The crux of the problem is to determine whether there are significant numbers of long-term survivors in those who have positive mediastinal lymph nodes removed, lymph nodes that would be left behind if a less extensive dissection had been performed. There are several patients in this category now and they will be reported later.

We cannot solve this by rhetoric; we cannot insist, or say with conviction, that the inclusion of lymph nodes is a worth-while addition to a lobectomy or a pneumonectomy, but by the same token no one can say we are wrong.

DR. JULIAN JOHNSON, Philadelphia, Pa.—The question was asked about blood vessel invasion in adenocarcinoma. It was 81 per cent in our series. It was 100 per cent in the undifferentiated tumors. The question was asked regarding a definition of operative mortality. If the patient died within 1 month of his last operative procedure, it was reported as an operative death.

I would like to point out that we quoted Watson's work so extensively simply because his were the best results we could find for radical pneumonectomy. I hope it is clear that it is not our desire to talk any group out of the use of radical pneumonectomy in the

treatment of cancer of the lung. We only wish to show that the thoracic surgeon need not feel obligated to perform a radical pneumonectomy as opposed to the standard resection for cancer of the lung, as judged by the 5-year survival data at present available.

DR. ALTON OCHSNER, New Orleans, La.—I am extremely happy to speak this morning for two reasons. First, because this session is dedicated to the memory of Evarts Graham. I was fortunate in being a senior student at Washington University when Evarts came to Washington as the new Professor of Surgery. Evarts and I became very close friends and he used to chide me a lot because I thought there was a very definite causal relationship between smoking and cancer of the lung. It was not until a number of years later that he became convinced that there was a relationship, and in his typically honest way he wrote to me and said, "I am going to have to eat crow. I think you are right."

This morning I would like to discuss some of the factors which might influence prognosis in bronchogenic cancer. (slide) Unfortunately, our results are not as good as those which have been reported. We have had 47 5-year survivals, and I would like to call attention to the fact that sex apparently plays a role. As you can see, 268 patients who were operated upon 5 years previously did not survive; the male-female ratio was 8:2, whereas in the 5-year survivors the male-female ratio was 4:8. So that, although carcinoma of the lung is relatively infrequent in women, when it does occur in women it offers a better prognosis.

Also, age in our series was of prognostic importance. Of the 5-year survivors, 38 per cent were less than 50 years of age, 61 or 62 per cent were between 50 and 70, and there were no 5-year survivors beyond the age of 70. In contrast, in the over-all group only 21 per cent were less than 50, 68 per cent between 50 and 70, and 10.5 per cent beyond 70.

The type of lesion, as someone mentioned, is a factor. Whereas 48.8 per cent of those not surviving had epidermoid carcinomas, 68 per cent of those who did survive for 5 years had epidermoid carcinomas, and the prognosis is worse in the adenocarcinoma group; only 8 per cent of all our 5-year survivors had adenocarcinomas.

(slide) It is a well-known fact that lesions of the lower lobe offer a worse prognosis and that they tend to metastasize. Until recently, these metastases were thought to be blood borne, particularly those of the suprarenal. Dr. Kenneth Meyer, one of my associates, working in the Laboratory of Anatomy at Tulane, has shown that there are definite lymphatic connections between the lower lobes of both lungs and the lymphatics below the diaphragm, to the suprarenal gland. This shows it in the right lung; this shows it in the left lung.

So there is a definite lymphatic connection between the lower lobes and the lymph nodes of the abdomen. We have had a total of 1,453 cases, of which 44 per cent were inoperable. There were 818 considered operable. We cannot talk these people into being operated upon as well as they can at Barnes Hospital; as you see 7 per cent of our patients refused surgery. There were 759 patients explored, which is 52 per cent of the entire group. There were 282, or 37 per cent of those explored, who were found to be nonresectable, with a 20 per cent mortality rate. That 20 per cent mortality does not represent the exploration but it represents the extent of the disease. There were 477 resected, which is a little less than one third, with again a 20 per cent mortality rate. In all the survival studies, the hospital deaths are included.

The resectability incidence has distressed me a great deal. You can see that from 1914 to 1956 there has been tremendous variation in resectability incidences. In 1944 we had our low resectability incidence of 18.8 per cent. It then increased, as one would expect, because with the information that cancer of the lung was a common disease and should be diagnosed more frequently, in 1948 we had an all time high of 47 per cent. Then there was an almost progressive downhill trend in resectability incidence until 1955 when we reached almost an all time low. This, I am sure, reflects the fact that about 12 years ago there was a great deal in our literature about viral pneumonitis, and this was succinctly

called atypical pneumonitis. But, unfortunately, the medical profession has come to think of atypical pneumonitis as being synonymous with viral pneumonitis, and I am sure that the decrease in the incidence of resectability is due to the fact that these patients were treated for months with a diagnosis of viral pneumonitis when in reality they had a bronchogenic cancer.

As I mentioned, age plays a role in all of our survivals; 38 per cent were under age 50, and 62 per cent were between 50 and 70.

Nodal involvement was also a factor. These are clinical laboratory investigations. Where there is evidence of extension beyond the lung at the time of operation, the surgeon designated it as a palliative procedure. I would like to call attention to the fact that in our series, 42.5 per cent had extension beyond the lung at the time of operation. So that, although these represented extensive disease that extended beyond the lung, this 42 per cent did survive 5 years.

Of the total series, 80 per cent had nodal involvement, 38 per cent had pleural involvement, and 6 per cent had distant involvement. I do not want you to get the impression that we operate upon people who have evidence of distant involvement; we do not. These were people in whom we felt there was no evidence of distant involvement, but who died, and autopsy showed that they did have distant involvement. Of the 5-year survivals, 34 per cent had nodal involvement, and 8.5 per cent had extension elsewhere to the pleura or to the chest wall.

Of our cases, 34 per cent of the survivors had nodal involvement and 66 per cent no nodal involvement.

(slide) This is the recapitulation of what I have given you. In those in which the lesion was limited apparently at the time of operation, 32 per cent are alive after 5 years; of all the resections, 15 per cent, and in those in whom there was extension beyond the lung of some type, 9 per cent. One must consider this in the light of the mortality rate in the general population; at this age only 85 per cent of these people in the general population are alive after 5 years. I feel that, after a period of 3 years, the decrease in the survival rate is almost entirely due to other causes rather than carcinoma of the lung. So I think one can say, as Dr. DeBakey and I called attention to a number of years ago when we made our first survival report—about 10 years ago, if a patient is alive and well at the end of 3 years, following extirpation therapy for bronchogenic cancer, his chance of living without evidence of disease is good. We have had 2 patients who have developed carcinoma in the opposite lung after their 5-year survival. These are the only 2 patients who did not discontinue smoking.

CORONARY HEART DISEASE AFTER TWENTY-FIVE YEARS

Claude S. Beck, M.D., Cleveland, Ohio*

IT IS an honor for me to present this paper on this part of the program designed to commemorate Dr. Evarts Graham. There is no other place more appropriate to honor Dr. Graham than at this meeting. It is our pleasure to think of him today and it is easy to imagine him sitting here in the first row of seats listening intently to everything that is said, with the speaker watching him to see how he is reacting.

Although he is not here, he will live with us for many years because he created so many memories that have enduring value. He had a positive dynamic mind, a restless curiosity, a strong body which enabled him to work and, in addition, he had the courage and the power to speak out for those things in which he believed—these were some of the qualities that made him the great surgeon that he was. His contributions are of such value that he will live long in the annals of medical history.

INTRODUCTION

The history of coronary heart disease, the world's greatest killer, may be divided into three periods identified as follows: (1) by the correct location of the disease, (2) by the electrocardiogram, and (3) by the recognition of the axiom for treatment. The fourth period, of questionable realization, is that of prevention and retardation of the disease.

CORRECT LOCATION OF THE DISEASE

This was accomplished in the 1912-1918 period[1] when the seat of the disease was moved from the stomach to the heart. This was done not without opposition from the medical profession. By present-day standards this should not have required deep thinking. In that period there were great internists and pathologists, and necropsy examinations were not uncommon. But then, as now, the medical profession knew so much that was not so. It was of elemental importance to find the correct location of the disease but this did not change treatment. "Therapy had not progressed much beyond that used during previous centuries, consisting almost exclusively of rest and sedation."[2] To this has been added the use of anticoagulants. There exists a strange complacency toward this disease. There is no relationship between treatment and

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Read at the Thirty-eighth Annual Meeting of The American Association for Thoracic Surgery at Boston, Mass., May 16-18, 1958.

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death. There is no responsibility for death. This futility has paralyzed the thinking and has created the belief that to do nothing more than the old routines is to make no mistake. Nowhere in medicine is there such intellectual emptiness as exists in the understanding and treatment of this disease.

THE ELECTROCARDIOGRAM

The electrocardiogram has disguised this vacuum and has given respectability to the knowledge of the subject. The electrocardiogram has risen to great heights in the erudition of this disease whereas blood supply to ischemic muscle is dwarfed into nothingness. This false relationship in values enabled the electrocardiogram to dominate the picture of the disease. It has even enabled it to pass as therapy. Many a patient believes it helps him because his physician takes so many of them. He knows they are expensive. Little does he know that they were not designed for treatment. Someone should speak out against unnecessary usage of the electrocardiogram and place it in its proper relationship to the augmentation of blood supply to ischemic muscle. Is this not an obligation of the medical cardiologist?

AXIOM OF THE DISEASE[3]

After a coronary artery is stenosed or occluded, the fate of the patient depends upon the amount of blood in the ischemic muscle formerly supplied by this diseased artery. Addition of blood preserves life; subtraction of blood destroys life. This is the elementary starting point for effective treatment and it has been almost entirely disregarded. It may be assumed that several of the medicaments, because they temporarily relieve pain, do temporarily provide blood to ischemic muscle. However, there has been little or no proof of this until recently when trinitroglycerine was tested in the author's research laboratory. Furthermore, there is no evidence to indicate that any of the medicaments change the anatomy of the crippled coronary circulation or that they make intercoronary communications. For long-term protection, therefore, medical therapy fails to develop collaterals which are the requirements of protection when either stenosis or occlusion of an artery develops.

PREVENTION AND RETARDATION

The contemplation of the period when arterial disease in the heart can be prevented or retarded produces an aura of greatness. If it can be prevented in the heart no doubt the same biologic principles will prevent it in the brain, extremities, and elsewhere in the body. Next to food, shelter, and the absence of war there is probably nothing more important. On a practical and realistic basis, however, prevention should not be confused with treatment. Between these two there is no relationship. The expectation of good things to come also contains the possibilities of slow progress or even failure. Expectation is no solace for the status quo in treatment. Society has a right to demand that methods for effective treatment be investigated now. Society has not been informed of the ineffectiveness of long-term medical therapy.

CONTRIBUTIONS FROM THE LABORATORY[4]

These are based on approximately 6,000 experimental operations on dogs in the past 25 years. The motivation of these experiments was to improve the crippled coronary circulation. While this was a guide in this work, physiologic concepts were also brought into being which explain the benefit produced by the experiments. Also the facts from the laboratory shed light on some of the clinical aspects of the disease. For example, the three death factors of the disease were given definition and the requirements for treatment of each death factor were brought under scientific scrutiny. The reversible nature of one of these death factors was defined and reversibility from death to life was accomplished in several human beings who had died from the disease.

Some of our experimental work has little or no application to treatment at the present time and this part of the work is omitted from this presentation. This includes the work on retrograde flow by placing a graft between aorta and coronary sinus. This approach protects the heart more than any other procedure that we developed, but it has certain side effects which are unfavorable and the operation of placing this graft is a difficult technical procedure. Important physiology was established by this work even though it may not have application to treatment of this disease at the present time. The work on the direct manipulation of the coronary arteries themselves, such as cleaning them out or placing shunts or grafts in them, is also omitted as impractical at the present time. These contributions from the laboratory are referred to briefly.

1. Blood supply to ischemic muscle is the axiom for treatment. When a ligature is placed around the origin of the descending ramus of the left coronary artery, 5 c.c. of blood per minute delivered to this occluded artery will preserve the heartbeat in the dog. Additional blood gives additional protection. Reduction has the opposite effect. This amount of blood does not support contractility of muscle nor does it prevent infarction, but in a large series of dogs it preserves the heartbeat. As stated above, this axiom is nothing less than the elementary starting point for effective therapy.

2. The electrical condition of the heart in relation to its oxygenated blood supply was investigated and four facts were established. In these experiments the heart was exposed and electrograms were taken from its surface by sweeping a cotton electrode moistened in sodium chloride solution across the ventricles. The following facts were established:[5]

a. A uniformly well-oxygenated heart is electrically stable. The pattern of the electrogram is the same from any part of the ventricles.

b. A uniformly and deeply cyanosed heart is electrically stable. Cyanosis is produced by clamping the intratracheal tube in the anesthetized dog. The pattern of the electrogram is the same as in "a." The cyanosed heart does not fibrillate. It stops in standstill. A blue baby has a stable heart. The facts referred to in "a" and "b" indicate that electrical

stability of the heart is independent of the amount of oxygenated blood delivered to it. Electrical stability is based upon a uniform distribution of the heart's own blood regardless of the amount of oxygen in this blood. In other words, stability is not related to oxygen content of muscle.

c. Ligation of a coronary artery produces an area of cyanosis in the muscle formerly supplied by this occluded artery. A blue or cyanosed island of muscle is produced in a pink or well-oxygenated heart. The S-T segment of the electrogram is elevated over the well-oxygenated muscle. It is depressed over the cyanosed muscle. This heart is electrically unstable and may fibrillate.

d. A coronary artery such as the descending ramus of the left coronary artery is cannulated. Into this artery, well-oxygenated blood is perfused. The intratracheal tube is clamped and the heart becomes cyanosed except the area supplied by perfused blood. This produces a pink, well-oxygenated area in a blue cyanosed heart. The electrogram is the same as in "c." The S-T segment is elevated over the well-oxygenated muscle and depressed over the cyanosed muscle. This heart is electrically unstable and may fibrillate. A baby with one coronary artery coming off the aorta and the other from the pulmonary artery has anginal pain and an unstable heart which may fibrillate. Two conclusions are established by experiments referred to in "c" and "d." One is that an uneven or checkerboard distribution of oxygenated blood produces electrical instabillity, fibrillation, and death, and these sequelae are produced by ligation of a coronary artery in a well-oxygenated heart or by delivery of well-oxygenated blood into a cyanosed heart. The heart electrocutes itself. The other conclusion concerns a long-standing physiologic concept that occlusion of an artery produces "current of injury." The placement of pink and blue muscle in juxtaposition does not require injury of muscle. The correct term for this is "electrical instability due to oxygen-differentials." Reduction in the degree of oxygen differential makes the heart electrically more stable. Increase in the degree of differential makes the heart electrically more unstable. Current of injury is an incorrect term and expresses an incorrect physiologic concept.

3. When a coronary artery is occluded two distinct and different biologic phenomena occur. One is electrical instability. The other is death of muscle (Fig. 1). One may occur without the other. Both may occur together. Thus, the heart can electrocute itself without infarct. Infarct, even successive infarcts, may develop without an electrical condition strong enough to fibrillate the heart. This separation is essential in order to understand the physiology of the disease and its clinical manifestations.

4. There are two approaches to effective treatment. Both of these conform to the axiom in Item 1.

a. Distribution of the heart's own blood so that a red cell, after it gets by the stenosis in the arteries, can go anywhere within the substance of the heart where it can do the greatest amount of good.

b. Addition of blood from outside sources, from grafts on the heart, by retrograde flow through veins, or by cleaning out the diseased arteries themselves or by other manipulations on these arteries. On the basis of considerable experimentation, the statement can be made that the heart resists the entrance of outside blood. It is not impossible to demonstrate arteries between graft and heart large enough to carry blood but their occurrence in experiments is infrequent. Perhaps better methods can be developed. Direct manipulation of the coronary arteries in my experience has not been successful.

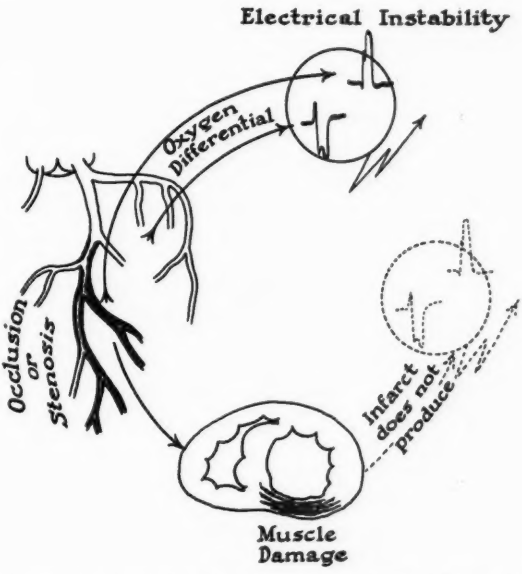

Fig. 1.—Two sequelae of occlusion or stenosis— (1) muscle damage, and (2) electrical instability. These responses are independent of each other. Viability of muscle is related to amount of blood supply; the electrical condition is related to distribution of blood. There is no conflict with the science when death occurs without infarct (as on the golf course) or in the presence of what the clinician refers to as a mild occlusion. The term mild is scarcely correct when it is associated with death and probably should not be used.

5. The amount of blood necessary to reduce the sequelae under Item 3 is in the range of several cubic centimeters per minute. In the human heart, as in the dog heart, there is always some blood in the ischemic muscle beyond the occlusion. The amount is variable and measures several cubic centimeters per minute. Quantities in the range of 2, 4, or 6 c.c. per minute added to that which is already present reduce the sequelae of occlusion. If this fact from the laboratory were properly appreciated, the opposition to the surgical approach in treatment would be reduced. Only the scientist in the laboratory appreciates the value of several cubic centimeters of oxygenated blood per minute delivered to ischemic muscle. This lack of understanding is, indeed, an unfortunate condition as it is fundamentally related to effective treatment of this disease.

6. Quantities of blood within this range are made available by the production of intercoronary arterial anastomoses and these can be produced by surgical operation. Several procedures contribute to their development. The author's operation consists of abrasion of the lining of the parietal pericardium and surface of the heart, narrowing of the coronary sinus to a diameter of 3 mm., a light application of 5 per cent aqueous solution of trichloracetic acid to the surface of the heart between coronary arteries, light application of powdered asbestos to the surface of the heart, and the application of parietal pericardium and mediastinal fat as a graft to the surface of the heart. Narrowing of the sinus and abrasion of the epicardium are omitted if the heart is irritable at operation.

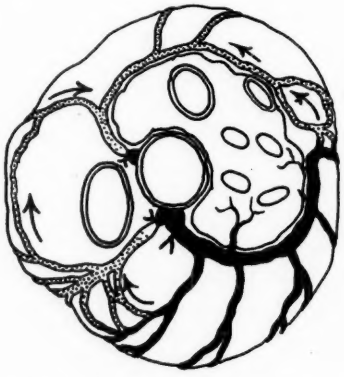

Fig. 2.—Sketch of Hahn-Beck heart. The artery in black is the circumflex ramus of the left coronary artery. It is the only coronary artery that carries in blood and this blood goes through an opening of 1 mm. in diameter. The descending artery and the right artery are occluded. These are stippled and they receive blood from the circumflex by way of intercoronaries indicated by the arrows. Once a red blood cell gets by the stenosis it can go anywhere in the substance of the heart where it is needed most.

7. Operation under Item 6 makes it possible to utilize the heart's own blood in the most effective way. It may not add new blood to the heart from outside sources but it does make more blood available to ischemic muscle from contiguous areas of muscle possessing a good blood supply. The average amount that it adds to ischemic circumflex muscle is 4.7 c.c. per minute or 282 c.c. per hour. The average amount that is already in the circumflex area of muscle after the artery is ligated in normal dogs is 3.8 c.c. per minute. After operation the average amount is 8.5 c.c. per minute. The average inflow into this artery in normal dogs is about 34 c.c. per minute.[6] The amount available after operation and subsequent occlusion, therefore, is approximately 25 per cent of the normal inflow. It is raised by operation from 11 per cent of the normal inflow to 25 per cent of the normal inflow. This is significant protection.

8. The normal coronary arteries are capable of delivering much larger quantities of blood than the amount that flows through the capillary bed. As

stenosis of the coronary arteries develops, the first conflict with normal physiology is produced by an uneven distribution of the blood that enters these diseased arteries. Distribution versus amount involves a fundamental concept in physiology. In the dog, all coronary inflow except that which goes through an opening of 1 mm. in diameter in one artery is enough to support the heartbeat and provide normal activity (Hahn-Beck dogs)[7] (Figs. 2 and 3). In the human being, an opening of 1.5 mm. in diameter can support the heartbeat. This blood must be evenly distributed in order to support the heartbeat. If it is not evenly distributed, the heart electrocutes itself. As the stenosing process becomes more severe, the second conflict with the physiology appears when there is not enough blood in the substance of the heart to support the heartbeat.

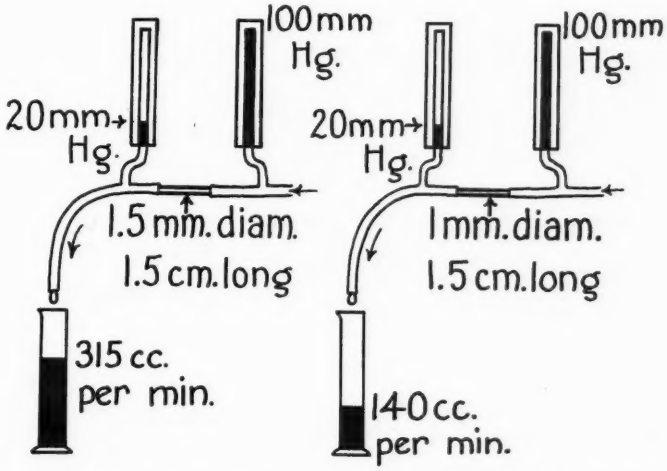

Fig. 3.—Rigid tube systems with stenosis. These flows are sufficient to maintain life. The dog has a flow of about 100 c.c. per minute through the capillary bed and the flow in the human being is about 250 c.c. per minute.

9. The operation under Item 6 has been well tested by the Mautz-Gregg backflow measurements,[8] by mortality after test artery occlusion, by infarct size in dogs that recover from test artery ligation, and by electrocardiographic evidence. The average backflow is increased 4.7 c.c. per minute or 282 c.c. per hour. The mortality rate is reduced when a major artery is occluded. For the descending ramus of the left coronary artery at its origin the reduction is from 70 per cent for normal dogs to 26.6 per cent for dogs in which the heart was previously protected by operation. The size of the infarct is reduced by 60 to 70 per cent. The electrocardiogram shows fewer and less severe alterations after artery occlusion in the protected dogs. A large amount of evidence indicating protection has been established.

10. There is still another fact concerning the value of several cubic centimeters of blood to ischemic muscle. A critical period exists when an

artery becomes occluded. If the heartbeat is maintained after occlusion for several days or for 1 or 2 weeks then intercoronary channels develop. It can be stated that they always develop after occlusion and they are of good size.[9] The blood made available to ischemic muscle by operation helps to maintain the heartbeat over the critical period so that nature has time to provide additional intercoronaries. This may be considered as an extra protection, so to speak, from the operation.

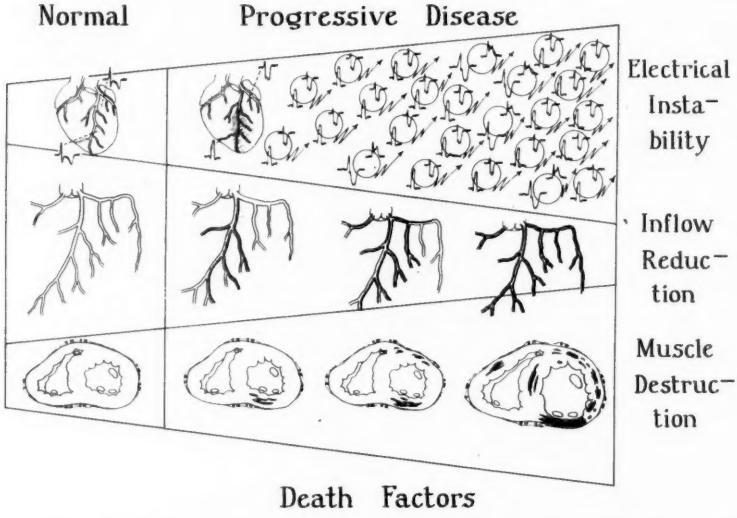

Fig. 4.—Three death factors in coronary heart disease—(1) self electrocution of the heart, (2) terminal stage in reduction of arterial inflow, and (3) terminal stage in destruction of muscle. Requirements for treatment of each factor can be determined.

THE INFLUENCE OF THE LABORATORY UPON CLINICAL CONCEPTS

The facts established in the laboratory are not only applicable to the treatment of the disease in the human being but they give some degree of order to the clinical understanding of the disease. Of fundamental importance are the three death factors in coronary heart disease (Fig. 4) and the combination of these factors as found in necropsy examinations (Fig. 5).

11. One factor of the disease that kills is by self electrocution of the heart. The majority of the deaths are due to this factor. It kills under a variety of conditions, on the golf course, when shoveling snow, when watching an exciting contest, during physical exertion, at rest, etc. In many instances the heart is too good to die. Sometimes this death factor is reversible and life can be restored.[10] The author actually accomplished this in 2 human beings who died from the disease and who are living several years later. If the profession were prepared, no doubt, restoration of life could be accomplished outside the hospital and a large number of lives might be saved.

The term "mild occlusion" is a clinical syndrome often described by the cardiologist. When a patient with a mild occlusion dies the terminology is wrong and a paradoxical situation is created. The term "mild occlusion" is used to refer to muscle damage without reference to the electrical condition that might assert itself. There should be no mystery in this association. Perhaps the term "mild occlusion" should not be used.

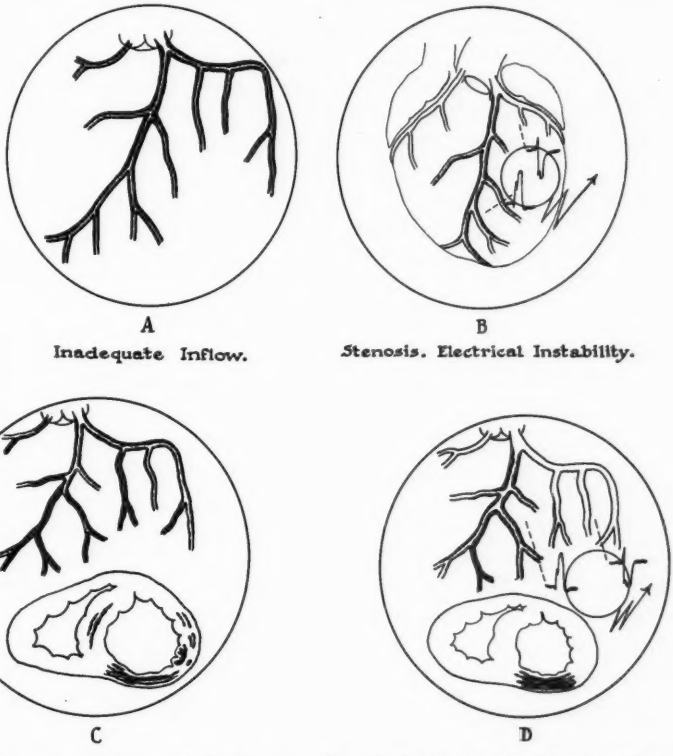

Fig. 5.—Various combinations of these factors are present in the heart. *A*, Inadequate inflow without infarct; *B*, self electrocution without infarct; *C*, muscle failure; *D*, self electrocution with infarct.

12. The incidence of self electrocution of the heart is reduced by the presence of intercoronary communications. This is probably the only death factor for which, at the present time, there exists effective surgical treatment.

13. Another death factor is inadequate inflow to support the heartbeat. The muscle may or may not be severely damaged. There is no effective operation for this condition at the present time. Clinical recognition of this condition is not always possible. Loss of strength and inability to get around may indicate severe inflow reduction.

14. The third death factor is loss of muscle. There isn't enough muscle to propel the blood and failure develops. The heart is enlarged. The only treatment is that for failure. There is no operation for this.

These 14 points convey the author's point of view concerning coronary heart disease. By design these points concern physiology and effective treatment. The origin and development of these points were slanted toward therapy. They break away from the established medical point of view as though a different disease is under consideration.

In this new design, the axiom for treatment displaces the electrocardiogram to a secondary position. The cleavage between the existing superstructure of the disease and this new design is comparable to the break that occurred in the 1912-1918 period. This new design is of equal or greater importance to making the correct location of the disease because it concerns effective treatment and there is no substitute for augmentation of blood supply to ischemic muscle.

EXPERIENCES ON HUMAN BEINGS[11]

The author's operation or some modification of it was done on 347 patients between Jan. 1, 1954, and Feb. 14, 1958. These were consecutive operations done at University Hospitals and Mount Sinai Hospital, Cleveland. Total mortality during the period of hospitalization was 21 or 6 per cent and the total mortality after discharge from the hospital during the period of somewhat more than 4 years was 32 or 9.2 per cent. Within this series were 100 consecutive operations with zero operative mortality, and in the subsequent series of 100 cases, there were 4 patients who died, making the mortality 2 per cent for 200 consecutive cases. The classification of salvage was applied to patients in whom the anatomic disease in arteries and/or myocardium was approaching the terminal stages. The inflow was approaching the critical stage when it would become inadequate for function and the destruction of muscle was approaching the stage when it would be inadequate to pump the blood. This classification of salvage cases was based upon an estimate made at the time of operation. In this group, there were 110 classified as salvage or 32 per cent; 234 were classified as nonsalvage or 67 per cent; and 3 were not classified. According to this classification, one third of the patients operated upon were approaching the terminal stages of the disease when mortality could be expected to occur in the not too distant future.

Many patients are not referred for operation until the disease approaches the terminal stages when the patient is told that medical therapy has nothing more to offer. In the 110 salvage cases, the total mortality during the 4-year period was 26 per cent—hospital mortality 12 or 11 per cent, subsequent mortality 16 or 15 per cent. In the 234 nonsalvage cases, the total mortality during the 4-year period was 10 per cent—hospital mortality 8 or 3 per cent, subsequent mortality 16 or 7 per cent. In the nonsalvage cases, the risk of operation is low, and the life span probably is excellent. In patients with coronary heart disease, this appears to be a good record and emphasizes the

desirability of early operation. The death factor in the early stages of the disease is the unstable electrical condition of the heart and operation has proved its value against this factor. The scientist accepts this as a statement of fact.

The heart was examined at operation according to a formula learned by experience. The size of the heart was noted. An enlarged left ventricle indicated myocardial damage. A rolling or rocking motion indicated that the muscle was not contracting in a uniform manner due to the presence of scar. Localized reduction or absence of pulsation indicated localized myocardial damage. Some of the scars did not come to the surface and could not be observed. The anterior and posterior aspects of the heart were observed. In some instances, the heart was irritable and could not be tipped up to examine the posterior surface. The arteries were examined. The common left artery was palpated. The circumflex artery and the right coronary artery cannot be observed or palpated. A soft pulsating common left artery indicated normal inflow. When this artery is soft and pulsating, the problem in therapy is not addition of blood to the heart, but distribution of what is there. A severely thickened or calcified common left artery indicates inadequate inflow. The problem then becomes one of addition of blood to the heart and this cannot be accomplished by surgical operation. The artery must be severely stenosed before inflow reaches terminal reduction. Beading and streaking with atheroma is observed in the descending ramus, its branches, and in the branches of the circumflex artery. Pulsation is observed to be normal, reduced, or absent. Irritability of the heart is noted. It sometimes occurs when the heart is manipulated but it was observed in 1 case when only the pericardium was picked up in a hemostat prior to opening it. The heart almost fibrillated when the pericardium was gently grasped in a hemostat. Indeed, grasping only the pericardial fat in a forceps almost fibrillated the heart. Marked irritability is a danger signal, and is considered an indication for a modified procedure such as the application of 5 per cent freshly diluted aqueous solution of trichloracetic acid to the surface of the heart between coronary arteries, and a light application of powdered asbestos to the surface of the heart. Marked irritability indicates severe inflow reduction. It is not necessarily associated with severe muscle damage. Indeed, the heart with extensive myocardial infarcts is often stable, probably due to the presence of intercoronaries which developed after arterial occlusion.

Examination of the heart at operation does not reveal all of the anatomic disease that actually exists in arteries and muscle. Some of these manifestations are found only at necropsy examination. The beating heart, however, gives some information not obtained by necropsy examination. The localized absence of pulsation and the outward bulge of an aneurysm are detected when the heart is beating. Any outward bulge of the heart, even though slight, was classified as an aneurysm. Analysis made at the time of operation is as follows: Size of the left ventricle, normal 221 or 64 per cent; enlarged, 126 or 36 per cent; aneurysm, 37 or 11 per cent; visible infarct,

250 or 72 per cent; no visible infarct, 97 or 28 per cent; arterial disease observed, 332 or 96 per cent; not observed 14 or 4 per cent; common left artery palpated, soft, and pulsatile, 261 or 76 per cent; thickened or calcified, 81 or 24 per cent.

According to this analysis, the hearts operated upon showed positive and usually severe manifestations of coronary heart disease. When a patient died and the heart was examined, the disease always was more severe than anticipated by the examination at the time of operation. When the heart stopped beating, complete recovery was possible in only 1 patient. This heart fibrillated 4 hours after operation and continued to fibrillate during a period of 35 minutes while the heart was being pumped by hand. This patient is alive more than 1 year after operation. In the other patients who died during operation, it was sometimes possible to restore the beat for periods of minutes or several hours, but always the heart went back into fibrillation. In some of these patients, the chest was opened, the heartbeat was restored, the chest was closed, the heart again fibrillated, the chest was opened, the beat was restored, and so on. One patient between these temporary deaths stated that she did not like the electric shocks, that she thought they were only for psychiatric patients. We worked on some of these patients as long as 16 hours and opened and closed the chest as many as four times. During these hours, the patients made conscious responses and understood what was stated. In several of these patients, an attempt was made to scrape out athermomatous deposits in the descending artery. This was never successful. In my opinion, this procedure has little to offer in treatment of this disease. When the common left artery is soft and pulsatile, the assumption is made that the inflow of blood into the heart is adequate and the problem in treatment is distribution of this blood rather than addition from other sources. The author's operation is designed to distribute this blood. Three out of 4 patients had a soft pulsatile common left artery and this was interpreted to mean a more favorable operative result.

A low operative mortality is necessary in order to make the operation acceptable. Coronary heart disease in any stage of its development kills as does no other disease. Death occurs with or without operation, but when associated with operation, then the latter assumes a causal relationship. In order to have a low operative mortality, it is necessary to be selective with patients for operation. Cardiac failure and marked enlargement of the heart are contraindications. Severe inflow disease is another contraindication. This occurs with or without muscle damage. Clinical recognition of this condition is often not possible, especially if the heart is of normal size, and there is little or no evidence of infarcts. Status anginosus and incapacitating pain may indicate severe inflow reduction, but some of the best clinical results were obtained in patients with incapacitating pain who tolerated operation satisfactorily. Recent activity of the disease, as indicated by a recent infarct or by an unusually severe attack of pain, is an indication to delay operation for several months, and 5 out of 8 patients in whom this was done, died during the waiting period.

In the past 2 years, the policy was established to withhold operation on patients whose blood pressure under anesthesia fell to 90 mm. Hg and did not spontaneously rise again without the use of drugs. This occurred in 14 patients in whom operation was not done and 3 of these died sometime later. Operation was refused to 15 patients because of severe disease determined after study in the hospital and 4 of these died subsequently. There is some danger in operating on any patient with coronary heart disease, but there is a group of patients in whom it can be stated before operation that the risk is slight. In the group with severe disease, but acceptable for operation, it is sometimes not possible to recognize those with marked inflow reduction. A "bad case" clinically may be a "good case" at operation and vice versa.

CLINICAL RESULTS

A survey of clinical results was completed recently. In this series of 347 patients there are 295 patients who are still living. Each of these patients was contacted by office visit, by telephone, or by questionnaire. The definition of excellent was applied to those patients who were completely or almost completely free of pain and took no medication after operation. The definition of good was applied to those patients who were improved after operation but had an occasional pain and took significantly less medicament. The vast majority were back to work. Of the entire group of living patients, 278 (94 per cent) received an excellent (32 per cent) or a good (62 per cent) result. In the group of 73 living salvage patients, the results were almost as good as in the entire group, with 22 per cent receiving an excellent result. These figures are such that the accuracy of the analysis might be questioned. They might appear to be too good and, therefore, not acceptable. These results were carefully compiled. It is possible for the operation to produce a biologic response in every patient in whom it is done and this in turn produces benefit in every patient operated upon. Another factor for consideration is that death removed some of those patients in whom the disease was progressing and this gave some selectivity to this group. Another factor, which is present whether the patient is treated by medical measures or by surgical operation, is the mental overlay associated with this disease. This factor is important but it should not be overrated. There probably are not many patients who, having gone through the discomfort of operation, would persist in saying they were better over a period of months or years if they were not better.

Many patients state that the anginal pain disappeared as early as 2 days after operation. These patients complain of incisional pain but state that the "old pain" has disappeared. This can be explained on the basis of improvement in the coronary circulation brought about by the inflammation on the surface of the heart occurring as early as several days after operation. The results in patients who had severe anginal pain before operation are almost unbelievably good. It is almost necessary to talk to these patients to appreciate the result.

There are several objective signs of improvement after operation. In 1 patient, the radial pulse on the left disappeared after coronary occlusion. This pulse was absent until operation was done 1 year later. On the second day after operation it was discovered that the radial pulse was present. Many patients state that they are warmer after operation. A study of skin temperatures of the feet was carried out and it was shown that if the feet were cold before operation, they became warmer after operation. The average rise in 11 patients was 7° C.[12] This is an objective (not subjective) change following operation.

Several other objective changes occurred after operation. In 11 patients, the chief complaint before operation was irregularity of the heartbeat due to ventricular premature beats or atrial premature beats. Several patients had episodes of sinus tachycardia.[13] These conditions were alarming to the patient. They improved or disappeared after operation. Ballistocardiograms were carried out on many of these patients. An abnormal ballistocardiogram was found in 10 out of 17 patients before operation. The ballistocardiogram in 8 of these 10 patients was improved after operation.[14] This was interpreted as improvement in the contractility of the myocardium and is a good correlation with the clinical improvement.

EFFECT OF GLYCERYL TRINITRATE (NITROGLYCERINE) ON BACKFLOW

Some of the drugs used in the treatment of coronary heart disease are being tested in the author's research laboratory by Dr. David S. Leighninger.[15] The same tests that were applied to the author's operation are being applied to these medicaments. So far, backflow determinations of the effect of nitroglycerine were done on 21 dogs. These tests were done as follows. The circumflex artery is ligated near its origin. The artery is transected distal to the ligature and a cannula is introduced into the artery. This cannula is connected to the carotid artery by a tube. Perfusion from this artery is stopped and the cannula is opened so that blood can escape from the distal circumflex artery. The mean aortic pressure is maintained at 100 mm. Hg. The blood that escapes is a measure of the amount of blood coming to the circumflex area of muscle by way of intercoronary arterial communications. The flow is measured for 30 seconds and is expressed in drops or cubic centimeters per minute. After a determination is made, the circumflex area of muscle is again perfused from the carotid artery. The backflow is determined by taking the average of several determinations made at 5-minute intervals. Nitroglycerine is then given intravenously and measurements are again made while the drug is effective. The mean increase in backflow was 0.8 c.c. or 12 drops per minute. The range in the experiments was 0.2 to 5.4 c.c. per minute. The duration of drug action was approximately 5 to 10 minutes.

These determinations are a measure of the most effective drug in the treatment of anginal pain. They are almost the only direct measurements of this drug-action on blood supply to ischemic muscle. It is indeed a strange commentary that this information, after years of usage on patients, originated

from a surgical laboratory by a surgeon who has had experience with this test. Without this experience, these positive measurements probably would have been missed or considered insignificant. The relief of pain is based upon 12 drops of blood per minute to ischemic muscle. The author's operation adds 4.7 c.c. per minute or almost 6 times this amount. The effect of this drug lasts 5 to 10 minutes. Then the backflow returns to normal. The increased backflow following operation is not temporary. It is permanent. These measurements probably place the effectiveness of operation in a new light.

CONCLUSIONS

1. A new design for the physiology and treatment of coronary heart disease has been created. This design concerns blood-supply to ischemic muscle and not the electrocardiogram.

2. Three death factors of coronary heart disease are defined. The requirements for treatment of each are given. One death factor is preventable. It is also reversible.

3. The important concepts of the disease are as follows:

a. The axiom.

b. Two approaches in treatment concern, (1) addition of blood to the heart, and (2) distribution of the blood that gets into the coronary arteries.

c. An uneven distribution of the heart's own blood is responsible for most of deaths from the disease and not an inadequate total inflow.

d. A uniformly cyanosed heart is electrically stable. The amount of oxygenated blood delivered to the heart is not a factor in electrical stability.

e. Well-oxygenated blood delivered into one coronary artery of a cyanosed heart can destroy the heartbeat. Red blood can kill the heartbeat under certain conditions.

f. A cyanosed area of muscle (as produced by arterial occlusion) in a well-oxygenated heart can destroy the heartbeat.

g. An uneven distribution of blood produces death by self electrocution.

h. The two sequelae of occlusion, (1) death of muscle and (2) electrical instability are not related.

i. Quantities of blood in the range of drops or several cubic centimeters per minute delivered to ischemic muscle are beneficial and protective.

j. Operation can produce intercoronary communications.

These concepts are poorly understood. It is the author's opinion that coronary heart disease is as poorly understood at the present time as were the bacterial diseases before Pasteur. The statement is sometimes made that "it is good to get your coronary occlusion and have it over." This statement is reminiscent of the expression "laudable pus" before Lister.

4. Nitroglycerine augments the amount of blood in ischemic muscle by about 12 drops of blood per minute in the dog. This drug effect lasts 5 to 10 minutes. It is suggested that the term *nitroglycerin unit* be used to relate these

laboratory measurements with the clinical effect of this drug. This unit, as such, combines clinical effect and quantity of blood and could be used in reference to other drugs and surgical operations.

5. The author's operation delivers several times this amount and this blood supply is permanent.

6. Results of operation are excellent or good.

If Evarts Graham were here he would have the mind to understand this work, and, second, he would have the courage and the spirit to fight for its application to patients. This work should not be allowed to remain as a second Semmelweis episode.

REFERENCES

1. Herrick, J. B.: Clinical Features of Sudden Obstruction of the Coronary Arteries, J. A. M. A. **59:** 2015, 1912.
   Herrick, J. B.: Angina Pectoris, J. A. M. A. **70:** 67, 1918.
2. Wright, Marple, Beck: Preface to Myocardial Infarction, New York, 1954, Grune & Stratton.
3. Beck, C. S.: The Development of a New Blood Supply to the Heart by Operation, Ann. Surg. **102:** 801, 1935.
   Beck, C. S.: Principles Underlying the Operative Approach to the Treatment of Myocardial Ischaemia, Ann. Surg. **118:** 788, 1943.
4. Beck, C. S., and Tichy, V. L.: The Production of a Collateral Circulation to the Heart, Am. Heart J. **10:** 849, 1935.
   Mautz, F. R., and Beck, C. S.: The Augmentation of Collateral Coronary Circulation by Operation, J. THORACIC SURG. **7:** 113, 1937.
   Stanton, E. J., Schildt, P., and Beck, C. S.: The Effect of Abrasion of the Surface of the Heart Upon Intercoronary Communications, Am. Heart J. **22:** 529, 1941.
   Beck, C. S.: The Coronary Operation, Am. Heart J. **22:** 539, 1941.
   Beck, C. S., and Mako, A. E.: Venous Stasis in the Coronary Circulation, Am. Heart J. **21:** 767, 1941.
   Beck, C. S.: Principles Underlying the Operative Approach to the Treatment of Myocardial Ischaemia, Ann. Surg. **118:** 788, 1943.
   Schildt, P., Stanton, E., and Beck, C. S.: Communications Between the Coronary Arteries Produced by the Application of Inflammatory Agents to the Surface of the Heart, Ann. Surg. **118:** 34, 1943.
   Beck, C. S., Stanton, E., Batiuchok, W., and Leiter, E.: Revascularization of the Heart by Graft of Systemic Artery Into the Coronary Sinus, J. A. M. A. **137:** 436, 1948.
   McAllister, F. F., Leighninger, D. S., and Beck, C. S.: Revascularization of the Heart by Vein Graft From Aorta to Coronary Sinus, Ann. Surg. **133:** 153, 1951.
   Eckstein, R. W., Hornberger, C., and Sano, T.: Acute Effects of Elevation in Coronary Sinus Pressure, Circulation **7:** 422, 1953.
   Beck, C. S., Leighninger, D. S.: Operation for Coronary Artery Disease, J. A. M. A. **156:** 1226, 1954.
   Beck, C. S.: The Old and the New in Coronary Artery Disease, J. Am. Geriatrics Soc. **4:** 315, 1956.
   Mautz, F. R.: Anatomical and Physiological Considerations in the Development of a Collateral Circulation to the Myocardium, Dis. Chest **31:** 243, 1957.
   Beck, C. S.: Coronary Artery Disease—Physiological Concepts—Surgical Operation, Ann. Surg. **145:** 439, 1957.
   Beck, C. S.: Coronary Artery Disease. A Report to William Harvey 300 Years Later, Am. J. Cardiol. **1:** 38, 1958.
5. Beck, C. S., and Leighninger, D. S.: Scientific Basis for the Surgical Treatment of Coronary Artery Disease, J. A. M. A. **159:** 1264, 1955.
   Brofman, B. L., Leighninger, D. S., and Beck, C. S.: Electric Instability of the Heart. The Concept of the Current of Oxygen Differential in Coronary Artery Disease, Circulation **13:** 161, 1956.
6. Brofman, B. L., and Beck, C. S.: Coronary Heart Disease. Hemodynamic Principles and Their Therapeutic Applications, J. THORACIC SURG. **35:** 232, 1958.
7. Hahn, R. S., and Beck, C. S.: Revascularization of the Heart: A Study of Mortality and Infarcts Following Multiple Coronary Artery Ligation, Circulation **5:** 801, 1952.

8. Mautz, F. R., and Gregg, D. E.: The Dynamics of Collateral Circulation Following Chronic Occlusion of Coronary Arteries, Proc. Soc. Exper. Biol. & Med. **36**: 797, 1937.
9. Zoll, P. M., Wessler, S., and Schlesinger, M. J.: Interarterial Coronary Anastomosis in the Human Heart, With Particular Reference to Anemia and Relative Cardiac Anoxia, Circulation **4**: 797, 1951.
10. Beck, C. S.: Fatal Heart Attack and Successful Defibrillation, J. A. M. A. **161**: 434, 1956.
11. Brofman, B. L.: Medical Evaluation of the Beck Operation for Coronary Artery Disease, J. A. M. A. **162**: 1603, 1956.
Antonius, N. A., Crecca, A. D., and Massarelli, L. G.: Clinical Evaluation of the Surgical Treatment of Thirty-Two Cases of Coronary Artery Disease, J. THORACIC SURG. **35**: 68, 1958.
12. Nevin, J. E.: Skin Temperatures of the Foot Before and After the Beck Operation for Coronary Heart Disease, To be published.
13. Brofman, B. L., and Bond, J. F.: Influence of the Beck Operation on Chronic Arrhythmias Associated With Coronary Heart Disease, To be published.
14. Brofman, B. L., Arbeit, S. L., and Adicoff, A.: Ballistocardiographic Evaluation in the Surgical Treatment of Coronary Heart Disease, Am. J. Cardiol. In Press.
15. Leighninger, D. S., and Beck, C. S.: Effect of Nitroglycerine and Other Drugs on Blood Supply to Ischaemic Myocardium, To be published.

DISCUSSION

DR. FREDERICK R. MAUTZ, Cleveland, Ohio.—It is a great privilege to discuss the paper of my good friend and long time colleague, Dr. Claude Beck, on this program honoring Dr. Evarts Graham.

I want to review briefly the physiologic basis for the Beck procedure. We believe that the dog heart provides information comparable in every way to the situation in the human heart. In the normal heart, the coronary arteries are pretty much end arteries but almost always show a considerable network of fine intercoronary arteriolar anastomoses.

When we isolate a major arterial ramus in a functioning heart as, for example, the circumflex branch, ligate and distally divide the artery, we note a little back bleeding of bright red arterial blood from the peripheral cut end. This blood has traversed the intercoronary anastomotic channels. On first appearance this seems to represent a very slight amount of collateral but, as we study a large number of preparations, we find a wide variation in the backflow. In a group of 52 normal dogs this varied from 0.1 c.c./min. to 22 c.c./min. In a comparable series of dogs that had previously had a Beck I procedure done, the backflow varied from 1 c.c. to 27 c.c./min., the average being twice that of the normal hearts. In both groups, this backflow represents a basic intercoronary collateral in hearts that have had *no previous arterial obstruction.*

If we add the factor of chronic arterial obstruction we find great increases in the backflow in the majority of experiments, on occasion exceeding 100 c.c./min. of arterial blood. Peripheral coronary pressures in such preparations occasionally approach aortic pressure whereas peripheral coronary pressure in the normal heart varies from $\frac{1}{14}$ to $\frac{1}{4}$ of mean aortic pressure. The increase has been shown to be due to enlargement of intercoronary arterial channels which have become capable of providing a functional collateral circulation from unoccluded coronary arteries.

The surgical procedures of the Beck I operation, namely, abrasion of the epicardium, narrowing of the coronary sinus, and the application of powdered asbestos, cause an increase in the basic intercoronary anastomoses and also aid in the natural process of gradual enlargement of anastomoses due to the factors of inflammation plus a mild degree anoxia due to reduced venous blood flow. The result is a more uniform distribution of arterial blood in the myocardium. Our studies seemed to indicate that extracardiac sources of blood are not tapped until quite late. Some recent studies of Lord and associates, of New York, using a method of extracorporeal circulation, report what I believe are the first direct measurements of extracoronary collateral, and obtained values as high as 10 per cent of the control coronary flow measurements.

I am confident that this door of Nature has now been opened. I do not believe we have reached the ultimate in our understanding of the phenomena with which we are

dealing. Let us further develop our principles of treatment and continue to be guided by the spirit of Dr. Graham in our quest of facts. We must study, measure, and prove the facts in the laboratory before we indiscriminately try to apply new procedures on man.

DR. A. VINEBERG, Montreal, Canada.—I wish to congratulate Dr. Beck on his wonderful work which has been carried out over a great many years, in spite of much opposition and terrific odds.

There now remains little doubt that partial ligation of the coronary sinus opens up intercoronary collaterals and provides better distribution of the blood that has managed to reach the heart muscle beyond the points of narrowing in the coronary arteries. However, there is absolutely no experimental evidence that extracardiac blood ever reaches an ischemic heart following the Beck I operation. Further, during all these years, no injected human specimens have been presented to show arteriolar-sized channels between pericardium and myocardium. The ever-changing character of coronary artery heart disease and the variations in the selection of patients for treatment make the evaluation of any method difficult. For this reason the ameroid method of experimentally constricting

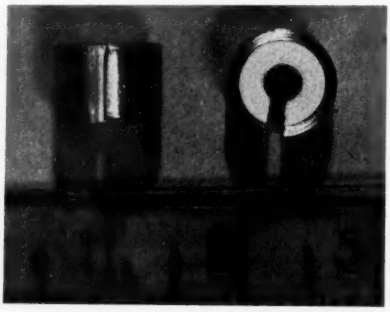

Fig. 1.—Photograph of casein ameroid with steel housing showing slot through which artery passes into central hole.

TABLE I. CORONARY ARTERY AMEROID CONSTRICTION TEST OF MYOCARDIAL REVASCULARIZATION PROCEDURES*

OPERATIVE PROCEDURE	NO. OF ANIMALS	DEAD UNDER 30 DAYS	SACRIFICED DUE TO ILLNESS	SURVIVED	
				4 WK. TO 4 MO.	6 MO. OR LONGER
Control	22	19 (86%)	2 (14%)	1	0
Beck I	10	9 (90%)	0	0	1 (10%)
Cardiopneumopexy (Harken)	10	9 (90%)	0	0	1 (10%)
Ivalon tube, aorta to ventricle, all grafts blocked	12	11 (92%)	0	0	1 (10%)
Internal mammary and subclavian artery ligation	8	8 (100%)	0	0	0
Internal mammary artery implantation only—of which 18 arteries were open	19	0	0	13 (68%)	6 (32% [open])
Internal mammary artery implantation + Coronary sinus ligation	10	0	0	3 (30%)	7 (70%)

*In all animals, petrolatum-coated ameroid constrictors were placed around anterior descending and circumflex branches of the left coronary artery.

coronary arteries has been developed in our laboratory. Constricting ameroids (Fig. 1) are placed around the origins of both the anterior and circumflex branches of the left coronary arteries. These slowly absorb water and gradually narrow the coronary vessels so that all control animals die under 30 days or have to be sacrificed, due to illness, within 6 weeks. The lumina of the coronary vessels are reduced to about 20 per cent of their

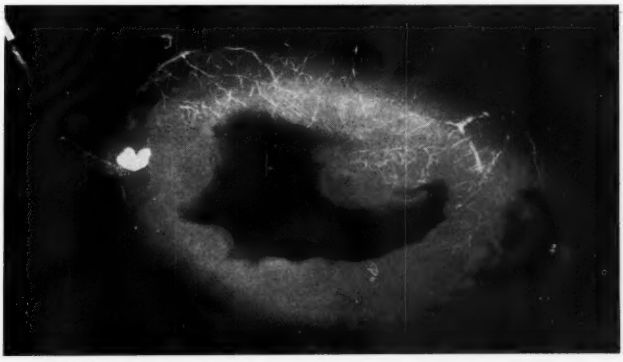

Fig. 2.—Filling anterolateral wall left ventricle by injecting Sclesinger mass into 2-month-old implanted internal mammary artery in animal which died from ameroid constriction of anterior descending and circumflex arteries.

Fig. 3. Fig. 4.

Fig. 3.—Filling of entire left ventricular coronary arteries through internal mammary artery placed in heart 6 months before in animal that survived ameroid constriction of anterior descending and circumflex arteries.

Fig. 4.—Animal 151. Angiocardiogram of left ventricle filled through 6-month-old internal mammary implant, showing mammary coronary anastomoses and filling of coronary arterioles. Anterior descending coronary artery completely blocked; circumflex coronary 90 per cent occluded.

normal size. Extensive intercoronary collaterals form, but not in sufficient size to prevent death. The control animals die suddenly during fights, after eating, or while exercising. With this standardized technique, it is possible to objectively evaluate methods of treating coronary artery insufficiency. It is quite clear that any procedure that prevents death and infarction when the two main branches of the left coronary artery are narrowed or occluded should be worth while. The surgical techniques tested by Dr. B. Mahanti this year are listed in Table I.

There were 22 control animals; the majority died under 30 days, 3 survived less than 6 weeks and were sacrificed due to illness.

It is interesting to note that the Beck I operation, cardiopneumopexy as done by Harken, and Ivalon tube grafts from aorta to myocardium failed to prevent death except in 10 per cent of a total of 32 animals. Internal mammary artery implantation alone, in 19 animals, provided some protection in 13 of the animals which lived 4 weeks to 4 months. Of these, there were twelve open internal mammary arteries, but mammary coronary anastomoses were limited to the anterior ventricular wall (Fig. 2). Of the remaining 6 animals, all the internal mammary arteries were open, all lived 6 months or more, could not be killed by exercise, and all showed extensive mammary coronary anastomoses involving the entire left ventricle (Fig. 3). When partial ligation of the coronary sinus was performed simultaneously with internal mammary implantation, 8 of 10 animals had open arteries. Three animals died 4 weeks to 4 months afterward but 7, or 70 per cent, lived 6 months or more. All internal mammary arteries were open and showed extensive mammary coronary anastomoses. Further, the left coronary arteriolar bed was satisfactorily filled through the internal mammary artery by angiocardiogram (Fig. 4). Since obtaining this experimental evidence, internal mammary artery implantation and coronary sinus ligation have been performed on 2 human beings.

DR. JOHN STRIEDER, Brookline, Mass.—Surgical approaches to chronic coronary artery disease have been a leading topic for many years. The treatment of coronary occlusion during the acute phase by removal of the obstruction to flow, and support of the circulation by extracorporeal pumping as a life-saving measure following massive infarction are two other aspects of the coronary problem that are likely to interest this Association for many more years.

The badly damaged heart will require support for days rather than for hours. In the Circulation and Surgical Research Laboratories at the Boston City Hospital we have been working on a veno-arterial pumping system without an oxygenator that we believe will be capable of providing such long-term support.

Blood is drained by gravity from the superior vena cava and pumped into a femoral artery. The coronary and cerebral systems are supplied by fully oxygenated blood from the lung. Superior caval blood, draining from the upper half of the body, is still 50 to 60 per cent saturated with oxygen. The lower extremities and splanchnic organs are able to function on this moderately reduced blood while relatively inactive. The pump is pneumatically operated with Hufnagel type ball-valves at each end, and is exceptionally gentle with blood elements.

It has been possible to pump as much as 70 c.c./Kg./min. with this equipment. In this slide, the lightly stippled columns are control cardiac outputs, the cross-hatched sections heart output during bypass, and the solid areas the bypass flow. The slide shows that the volumes pumped amount to about one half the body requirement. The output of the heart is reduced by approximately the volume pumped.

This slide shows the effect of veno-arterial pumping on aortic pressures. Pumping raises the diastolic pressure, a factor that should favor better flow through the heart muscle itself while the volume component of heart work is reduced.

We have been able to pump 50 to 60 c.c./Kg./min. continuously for as long as 54 hours with survival and only transient, minor effects on the clotting mechanism, acid-base

balance, and electrolyte pattern. We believe the hemodynamic effects of such veno-arterial pumping for a period of days will prove to be beneficial following major infarction of heart muscle.

DR. DONALD MULDER, Los Angeles, Calif.—Since Dec. 1, 1957, Dr. William Longmire and Dr. Jack Cannon of the UCLA Surgical Department have operated upon 5 patients for severe incapacitating angina pectoris. Previous studies of Blumgart and Schlessinger, Gregg, Thornton and Mautz, as well as numerous autopsy observations, have led us to believe that localized occlusion of the major coronary arteries with a patent distal arterial tree occurred frequently in the patient with angina pectoris without evidence of myocardial infarction or generalized atherosclerosis. In an especially devised exercise test, none of these patients could exercise for more than 3½ minutes without typical pain and the electrocardiographic changes of myocardial ischemia.

At operation one or more, usually two, of the major coronary arteries were found to be almost completely occluded. A technique of endarterectomy was perfected in the dog and found readily applicable in the human being. In the 5 cases mentioned, the obstructing plug was removed from both the right main and the left anterior descending coronaries in 2 patients. In all cases, the smaller distal branches were patent by inspection, palpation, and by demonstration of adequate "back bleeding" once the obstructing core was removed from the artery and its major branches. The technique is as follows:

The results of this procedure have been quite encouraging. Although the second patient was lost from asystole after the removal of the core from the second major coronary vessel, the other 4 patients have all survived. They have been entirely free from angina for the relatively short period since surgery. Exercise tolerance has been greatly improved in all patients and the ECG tracings do not show evidence of myocardial ischemia. We believe further evaluation of the technique is indicated as the early experiences are most promising.

SIR RUSSELL C. BROCK, London, England.—Although I have had the privilege of visiting the United States on many occasions, I am ashamed to say this is the first time I have been able to attend a meeting of The American Association for Thoracic Surgery. It is a particular source of pleasure to me to be present on this occasion and to pay tribute to Dr. Graham. I do not wish to add to the eulogy so ably presented by Dr. Burford this morning, but I would like to say that I feel this tribute very deeply because I had the privilege of being one of Dr. Graham's "boys," having worked with him in 1930-31. One thing that was mentioned, and very rightly so, was the great friendship that Dr. Graham encouraged between England and this country through the plan of inviting trainees here. And that brings up another point I would like to mention, Sir: I was very happy to see you wearing the chain of office which was presented last year by the Association of Thoracic Surgeons of Great Britain and Ireland to this Association as a bond of friendship between the two Associations. I am happy to see also that not only does the badge of office grace the President, but that the President graces the badge.

I should like to take as my theme the fact that Dr. Claude Beck has presented his observations based on a quarter of a century of work. The whole world looks to the United States for its achievements with regard to coronary artery disease. As you know, O'Shaughnessey of London in the early 1930's did some basic work on cardio-omentopexy, but since that time the greater part of the work has emanated from the North American continent. I was at first rather reluctant to speak on this subject because on a recent occasion I made some comments which Dr. Beck misunderstood but I am glad to have the opportunity to clarify my remarks. I have been reluctant until recently to embark upon these various surgical procedures for the improvement of coronary artery disease because, quite frankly, I have been unimpressed with the results of the attempts to bring a new blood supply to the heart from outside. I would say that the position is justified. Dr. Beck's own conclusion as a result of his work is that the heart seems to resist the

bringing of a blood supply from the outside. I am much impressed with Dr. Beck's presentation, not of the ability to bring a new blood supply from the exterior, but of the redistribution of coronary blood flow within the myocardium by increase in intercoronary anastomoses. This seems to me to be a logical and accepable result of the various stimulating factors caused by the Beck procedure and can well explain the good clinical results obtained.

That the simple act of opening the pericardium and examining the heart manually can be followed by quite profound reaction and disturbances is well known to all of us and reaches its most notable form in the so-called "post-valvotomy" syndrome, which is really a "postpericardial insult" syndrome and can follow simple needle puncture of the heart. It ordinarily has nothing to do with recurrence of rheumatic activity.

The effects of a cardiotomy are well known to any cardiac surgeon who has reoperated on the heart within a year of the first operation. The myocardium is soft, often discolored, and the patient's reaction to the interference may be unduly severe. It seems as if the heart easily suffers a notable traumatic myocarditis and to me it is easy to understand how such a process results in a great increase of intercoronary anastomoses.

When I say that I am unconvinced that a new and effective blood supply can be brought to the heart from outside, I do not mean that I am criticizing adversely the many splendid attempts to revascularize the heart from a systemic source. I merely state that so far I am not convinced that they succeed in the way they claim to succeed, although they may bring benefit by some other mechanism. I just do not want, today, to become involved in a critical discussion of these various procedures. I applaud the spirit and purpose which guides them and hope that they will achieve success.

DR. BECK (Closing).—There are two approaches for effective treatment. One involves addition of blood to the heart. On the basis of a large experience, the statement is made that the heart resists the addition of blood from outside sources. Many years ago my associates and I grafted tissues on the heart including the internal mammary artery with the expectation that blood would go across the graft to the heart. Later on, the specimens were injected with a watery dye and routinely it was possible to inject from the heart into the graft and from the graft into the heart. But, when we used a thicker material consisting of a solution of barium sulfate and gelatin, it was only the occasional specimen that showed blood vessels large enough to carry blood. Several specimens injected with this material entered the internal mammary artery which was grafted on the heart (for specimens see Mautz and Beck, J. THORACIC SURG. **7**: 113, 1937, and Beck, Ann. Surg. 118: 792, 1943). I have also seen specimens in Dr. Vineberg's laboratory in which channels large enough to carry blood were present. But, in the laboratory, we and others have had indifferent success in demonstrating such vessels. Perhaps we did not do our experiments right. We have also added blood into the venous system, directing it to the capillaries in a retrograde direction. This worked well for awhile but later on intimal thickening cut off this inflow, but this operation gave the heart an excellent set of intercoronaries and, indeed, the greatest degree of protection was provided by this operation.

Early in our work (1932) the idea of cleaning out coronary arteries was considered and applied first in autopsy specimens and later on at the operating table after the death of the patient. The possibility of yield seemed to be small. This idea is being revived and is being tried on patients even though surgeons have not been successful in arteries of this size elsewhere in the body. The widespread involvement of the coronary tree by the disease, often including secondary branches of coronary arteries in a large percentage of cases, the possibility of changing stenosis into occlusion by the surgical manipulation, and the possibility of creating an imbalance and electrocuting currents are considerations that will determine success or failure of this manipulation. Dr. John Madden of New York told me of his success in cleaning out the descending artery but at autopsy the right and circumflex arteries were severely stenosed. It is possible that this created an imbalance, oxygen differentials, and electrocuting currents.

I am grateful to Sir Russell Brock for his discussion; I appreciate it. He emphasizes distribution of the heart's own blood as one method of attack in treatment. I am pleased that he accepts this as a possibility for effective treatment. I had the privilege to attend the Harvey Tercentenary Congress in London last year. I shall never forget this meeting. In grace, dignity, and historical significance, this meeting could not be duplicated anywhere else in the world except in London and for William Harvey. The physicians of Britain have warm affection for The American Association for Thoracic Surgery. They showed it last year when the President of their Association, Mr. George Mason, presented to our President, Dr. Cameron Haight, a medallion to establish the Chain of Command to be passed on from President to President for all time into the future.

THE CLINICAL SIGNIFICANCE OF COR PULMONALE IN THE REDUCTION OF CARDIOPULMONARY RESERVE FOLLOWING EXTENSIVE PULMONARY RESECTION

Robert W. Harrison, M.D. (by invitation), William E. Adams, M.D., Edwin T. Long, M.D. (by invitation), Benjamin Burrows, M.D. (by invitation), and Arthur Reimann, M.D. (by invitation), Chicago, Ill.

SINCE the first successful one-stage pneumonectomy was performed over 25 years ago there has been great interest in the alterations of respiratory and circulatory function following the extensive loss of lung tissue.

Longacre[1, 2, 3] and his associates in experimental studies on dogs observed the effects of pneumonectomy upon pulmonary and cardiocirculatory functions in young growing and adult animals. In puppies subjected to pneumonectomy, enlargement of the remaining lung and histologic evidence of pulmonary hyperplasia were reported. The capacity of these animals to perform exhausting exercise was comparable to normal control animals. Similar studies on adult animals revealed findings of emphysematous change in the remaining lung in the form of fragmentation of alveoli and loss of elastic tissue. These animals showed marked physiologic deficiencies under conditions of physical stress.

Subsequently, clinical studies of the effects of pneumonectomy in children were reported. In a study of 4 young children followed up to adult life, Lester, Cournand, and associates[4, 5] observed that 2 of the patients, in spite of a moderate degree of pulmonary distention, showed a reduction in breathing reserve from normal and a marked degree of oxygen unsaturation in the arterial blood during strenuous exercise. The other 2 patients showed improved adaptation and good physiologic performance. A study of 10 children by Peters and associates[6] revealed that in spite of increases in size of the remaining lung with high residual volumes in one half of the cases, the physiologic responses of these patients to exercise were within the range of normal.

Observations on adults following pneumonectomy have also been reported. Cournand and Berry,[7] in a study of 12 patients before and after pneumonectomy, found the chief difference between postpneumonectomy patients and normal subjects was the reduction in breathing reserve in various states of activity. This reduction was caused by a decrease in maximum breathing

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capacity and was greater in the older patients. It was their impression that "the late effects of pneumonectomy upon gas exchange in the lungs, the state of respiratory gases in the arterial blood and the cardiocirculatory function were insignificant." In a later study on the pulmonary circulation and alveolar ventilation perfusion relationships after pneumonectomy, Cournand and co-workers[8] demonstrated that in 9 patients studied late after pneumonectomy there was an increase in the size of the remaining lung, a reduction of maximum breathing capacity, an approximately normal arterial oxygen saturation at rest and slightly below normal during exercise; the average pulmonary blood flow and mean pulmonary arterial pressure were within the normal range at rest and that a mild pulmonary hypertension inevitably developed in the course of mild exercise. Pulmonary function studies of Birath[9] and Burnett[10] and their associates produced findings similar to those of Cournand.

It has been recognized for some time that in certain individuals, notably those in the older age group, extensive pulmonary resection (pneumonectomy) with an immediate reduction of cardiorespiratory reserve results in cor pulmonale, right heart failure, and death.[11, 12] However, more recently interest has been aroused by a group of postpneumonectomy patients who were able to adjust to the immediate effects of pneumonectomy and resume their work and activities without disability; only years later to begin to experience progressive dyspnea on exertion resulting in a marked reduction of their functional capacity and sometimes forcing the individual into a state of retirement and semiinvalidism. The "late" effects experienced by these individuals were not always well correlated with abnormalities in their pulmonary function studies. Occasionally, such a patient would present the findings of right heart failure. Therefore, the mechanism of cor pulmonale resulting from a reduction of pulmonary parenchyma came under consideration. In view of the widespread use today of pneumonectomy as the operation of choice for carcinoma of the lung, as well as the frequent performance of extensive bilateral pulmonary resection, these "late" results should become of ever-increasing importance.

In an attempt to understand better the mechanisms involved in the "late" results of extensive pulmonary resection, and to elucidate further the role of pulmonary hypertension in such cases, physiologic studies were performed on a series of patients at various intervals following pneumonectomy and bilateral resection.

MATERIAL FOR STUDY

Physical characteristics and clinical data concerning the 29 patients studied are tabulated in Table I. There were 6 females and 23 males ranging in age from 23 to 74 years.

Pneumonectomy had been performed in 25 of the 29 cases; in 19 cases for a diagnosis of primary carcinoma of the lung, in 2 cases for tuberculosis, in 2 cases for cystic disease of the lung, in 1 case for chronic suppurative disease, and in 1 case for a bronchial adenoma. Two of the pneumonectomy patients

TABLE I. PHYSICAL CHARACTERISTICS, DIAGNOSIS, AND FUNCTIONAL CAPACITY OF PATIENTS FOLLOWING EXTENSIVE PULMONARY RESECTION

	PATIENT	AGE	SEX	SURFACE AREA M.^{2}	TYPE OF SURGERY	DIAGNOSIS	YEARS AFTER SURGERY	FUNCTIONAL CLASS
1	E. B.	44	F	1.73	Right pneumonectomy	Tuberculosis	3	II
2	B. B.	52	F	1.56	Left lower lobectomy, lingulectomy, and right middle lobectomy	Lipoid pneumonia	2	II
3	H. Be.	23	M	1.84	Left pneumonectomy	Cystic disease	5	I
4	H. Bo.	69	M	1.87	Left pneumonectomy	Carcinoma	3	IV
5	P. C.	69	M	1.49	Left pneumonectomy	Carcinoma	12	II
6	E. C.	42	M	1.88	Right pneumonectomy	Carcinoma	6	II
7	S. D.	65	M	1.84	Left pneumonectomy	Carcinoma	9	I
8	B. F. (1)	42	F	1.53	Right pneumonectomy	Bronchial adenoma	5	II
9	B. F. (2)	45	F	1.58	Right pneumonectomy	Bronchial adenoma	7½	II
10	J. G.	39	F	1.67	Right middle and lower lobectomies; left lower lobectomy and lingulectomy	Bronchiectasis	11	I
11	M. H.	68	M	1.90	Left upper lobectomy	Carcinoma	2	III
12	E. J.	49	M	1.62	Right pneumonectomy	Carcinoma	7	I
13	J. K.	37	F	1.77	Left pneumonectomy	Tuberculosis	3	I
14	E. K.	45	M	2.04	Right pneumonectomy	Carcinoma	6	II
15	A. L. (1)	46	M		Left pneumonectomy	Carcinoma	1 mo.	III
16	A. L. (2)	47	M		Left pneumonectomy	Carcinoma	1½	III
17	O. L.	54	M	1.79	Right pneumonectomy	Carcinoma	11	III
18	H. M. (1)	47	M	1.78	Right pneumonectomy	Carcinoma	3	I
19	H. M. (2)	49	M	1.64	Right pneumonectomy	Carcinoma	5	I
20	J. M.	57	M	1.90	Right pneumonectomy	Carcinoma	½	III
21	A. M.	53	M	1.64	Left pneumonectomy	Carcinoma	1	III
22	R. N.	72	M	2.00	Left pneumonectomy	Carcinoma	12	II
23	M. O.	63	M	1.84	Right pneumonectomy	Cystic disease, infected	3	II
24	L. R. (1)	61	M	1.71	Right pneumonectomy and thoracoplasty	Carcinoma	14	II
25	L. R. (2)	63	M	1.74	Right pneumonectomy and thoracoplasty	Carcinoma	15½	III
26	W. R.	58	M	1.74	Right pneumonectomy	Lung abscess	3½	I
27	E. S.	30	M	1.80	Left lower lobectomy and lingulectomy; right lower lobectomy	Bronchiectasis	11	I
28	J. So.	65	M	2.08	Right pneumonectomy	Carcinoma	3	II
29	J. Sz.	74	M	1.97	Left pneumonectomy	Carcinoma	7½	IV
30	H. T.	69	F		Right pneumonectomy	Carcinoma	3 wks.	IV
31	J. T.	64	M	1.59	Right pneumonectomy	Carcinoma	3½	II
32	W. W.	52	M	2.08	Right pneumonectomy	Carcinoma	3	II
33	P. W.	57	M	1.80	Left pneumonectomy	Carcinoma	7½	I

had also had a supplemental thoracoplasty performed on the side of the pneumonectomy. Four of these patients were studied on two occasions with an intervening period of 1½ to 2½ years.

Three patients had undergone bilateral pulmonary resection—2 for bilateral bronchiectasis and 1 for bilateral lipoid pneumonia. One patient had undergone only a left upper lobectomy for a primary carcinoma of the lung.

The studies were performed from 3 weeks to 15½ years after the surgical procedures.

Initially, investigations were made on patients who presented themselves with complaints of progressive dyspnea on exertion and consequent reduction of work and exercise capacity. Later, as interest in this problem was stimulated, the study was extended to include other patients who, after a comparable type of surgery, were entirely asymptomatic or offered only minor complaints. Their findings were compared with those in the group of symptomatic patients. The distribution of the patients as to age and type of surgery performed is summarized in Table II.

TABLE II. 33 PATIENTS
AGE OF PATIENTS—PROCEDURE PERFORMED

	20-29 YEARS	30-39 YEARS	40-49 YEARS	50-59 YEARS	60-69 YEARS	70-79 YEARS
Left pneumonectomy (11)	1	1	2	2	3	2
Right pneumonectomy (18)	0	0	8	4	6	0
Bilateral lobar resections (4)	0	2	0	1	1*	0

*Left upper lobe only.

METHOD OF STUDY

The patient undergoing investigation was hospitalized for 36 to 48 hours. During this hospitalization a complete clinical history and physical examination were obtained; electrocardiogram, chest roentgenogram, pulmonary function studies, and cardiac catheterization studies were performed. The clinical evaluation of the individual was carefully standardized by the use of a detailed questionnaire and clinical evaluation sheet.

The patient's functional capacity was graded on a scale of I to IV. Class I included those patients having no limitation of physical activity and in whom ordinary physical activity caused no discomfort; Class II signified a slight to moderate limitation of physical activity; Class III included those with a moderate to great limitation of physical activity; while Class IV was assigned to the patients unable to carry on any physical activity without discomfort.

Pulmonary function studies were carried out in the pulmonary physiology laboratory and included spirometric determination of ventilatory capacity, residual volume, nitrogen washout curves, ear oximetry and, in a few subjects, pulmonary diffusing capacity. Residual volumes were determined by a modification of the Darling method.[13]

Cardiac catheterization was employed to measure right heart pressures and cardiac output at rest and during moderate exercise. Pulmonary artery catheterization was not technically possible in all cases, and consequently only right

ventricular pressures were obtained in those cases. Arterial oxygen saturation was continuously recorded during the cardiac catheterization by a Wood earpiece and recording oximeter.[14] Right heart pressure measurements were made by means of a Statham strain gauge manometer and a Grass amplifier-recording instrument. Cardiac output was determined by the direct Fick method on 12 of the patients in a resting state and during the last minute of a 3-minute exercise period. Simultaneous mixed venous and systemic arterial blood samples were analyzed for oxygen content by the Van Slyke method.[15]

A 3-minute period of moderate exercise was required of each patient in the supine position by bicycling against resistance or flexing each leg against a 5-pound resistance.

RESULTS

The functional capacity of each patient at the time of study as determined by the history and clinical evaluation is tabulated in Table I. There appeared to be a rough correlation between the age of the patient at the time of the study and his functional capacity. However, a more striking relationship was found between the age of the patient at the time of his surgery and his resultant functional class (Table III); the older the patient at the time of surgery, the more likely he was to experience a reduction of functional capacity later.

TABLE III. RELATIONSHIP OF PATIENT'S AGE AT SURGERY AND RESULTANT FUNCTIONAL CLASS

FUNCTIONAL CLASS	10-19	20-29	30-39	40-49	50-59	60-69
	(AGE IN YEARS)					
I (11 patients)	2	1	1	4	3	0
II (12 patients)	0	0	4	2	2	4
III (7 patients)	0	0	0	4	2	1
IV (3 patients)	0	0	0	0	0	3

This was especially true of the group over the age of 60 at the time of surgery; in our series none of these patients was functionally unimpaired, and all 3 of our Group IV patients fell into this age group. There were no striking differences between the functional capacities of the right and left pneumonectomy groups, but the patients having undergone extensive bilateral resections presented little or no functional incapacity (Table IV). No relationship was demonstrated between the length of survival since pulmonary resection and the resulting functional capacity of the individual.

TABLE IV. 33 PATIENTS
PROCEDURE PERFORMED—FUNCTIONAL CAPACITY

	I	II	III	IV
Left pneumonectomy (11)	4	2	3	2
Right pneumonectomy (18)	4	10	3	1
Bilateral lobar resections (4)	2	1	1*	0

*Left upper lobe only.

TABLE V. PULMONARY FUNCTION STUDIES OF PATIENTS FOLLOWING EXTENSIVE PULMONARY RESECTION

CASE NO.		FUNCTIONAL CLASS	T.L.C. (L.)	V.C. (L.)	R.V. (L.)	$\frac{\text{R.V.}}{\text{T.L.C.}} \times 100$ (%)	MMF (L./SEC.)	MBC (L./MIN.)	1 SECOND V.C.	N_2 WASHOUT CURVE
Left Pneumonectomy										
3	H. Be.	I	5.2	3.95 (91%)	1.25	24	2.4	110 (82%)	71%	normal
7	S. D.	I		2.40 (72%)			1.1	69 (72%)	76%	normal
13	J. K.	I	3.85	2.05 (68%)	1.5	39	1.5	57 (60%)	74%	normal
33	P. W.	I	3.9	2.0 (57%)	1.9	49	1.5	63 (62%)	78%	normal
5	P. C.	II	3.2	1.8 (59%)	1.3	41	1.2	30 (40%)	69%	normal
22	R. N.	II	4.2	2.2 (63%)	2.0	48	0.56	30 (30%)	54%	abnormal
21	A. M.	III	3.4	1.6 (47%)	1.65	49	0.92	35 (37%)	70%	normal
4	H. Bo.	IV	3.8	1.05 (31%)	2.4	63	0.57	*	74%	slightly abnormal
29	J. Sz.	IV	5.66	2.5 (75%)	3.03	54	0.55	32 (43%)	46%	abnormal
Right Pneumonectomy										
12	E. J.	I	3.2	2.2 (59%)	1.0	32	2.4	59 (61%)	89%	normal
18	H. M. (1)	I	3.5	2.15 (57%)	1.35	38	0.79	54 (49%)	69%	normal
19	H. M. (2)	I	3.9	1.6 (42%)	2.1	54	0.6	31 (31%)	63%	normal
26	W. R.	I	3.08	1.6 (45%)	1.35	44	2.2	53 (57%)	98%	normal
1	E. B.	II	2.4	1.5 (54%)	0.9	37	0.55	36 (33%)	62%	normal
6	E. C.	II	3.6	2.3 (56%)	1.3	36	1.7	54 (44%)	81%	normal
8	B. F. (1)	II		1.70 (62%)				49 (55%)	85%	normal
9	B. F. (2)	II	3.2	1.8 (67%)	1.4	44	1.6	31 (39%)	70%	normal
14	E. K.	II	3.8	2.6 (64%)	1.2	32	1.6	63 (49%)	77%	normal
23	M. O.	II	4.1	2.1 (62%)	1.85	45	1.7	76 (79%)	80%	normal
24	L. R. (1)	II		1.03 (29%)			0.71	21 (23%)	79%	normal
28	J. So.	II	3.55	2.0 (57%)	1.45	41	3.0	50 (46%)	88%	normal
31	J. T.	II	3.75	1.55 (45%)	2.2	59	1.2	33 (39%)	78%	abnormal
32	W. W.	II	3.8	2.5 (64%)	1.3	34	1.7	65 (52%)	70%	normal
17	O. L.	III		0.97 (26%)			0.5	13 (12.5%)	50%	abnormal
20	J. M.	III	3.15	1.7 (46%)	1.35	43	1.5	56 (52%)	89%	normal
25	L. R. (2)	III	2.8	1.2 (34%)	1.6	57	0.52	31 (34%)	71%	normal
Bilateral Resections										
10	J. G.	I	4.0	2.0 (68%)	2.0	50	0.75	25 (28%)	58%	slightly abnormal
27	E. S.	I	5.1	3.5 (88%)	1.6	31	2.5	103 (79%)	77%	normal
2	B. B.	II		1.48 (58%)				23 (32%)	67%	
Left Upper Lobectomy										
11	M. H.	III	4.3	1.9 (56%)	2.4	56	1.5	54 (57%)	82%	normal

*Unable to perform test.

T.L.C. = total lung capacity; V.C. = vital capacity; R.V. = residual volume; MMF = maximum midexpiratory flow; MBC = maximum breathing capacity; 1 sec. V.C. = per cent of vital capacity expired in first second; N_2 Washout Curve = nitrogen washout curve.

The results of the pulmonary function studies and cardiac catheterization studies are tabulated in Tables V and VI and are represented graphically in Figs. 1 and 2. The average results will be discussed separately for each functional class.

TABLE VI. CIRCULATORY STUDIES OF PATIENTS FOLLOWING EXTENSIVE PULMONARY RESECTION

CASE NO.	FUNCTIONAL CLASS	RIGHT HEART SYSTOLIC PRESSURE (MM. HG)		ARTERIAL OXYGEN SATURATION (%)		OXYGEN CONSUMPTION (ML./MIN.)		CARDIAC OUTPUT (L./MIN.)		CARDIAC INDEX (L./MIN./M.²)	
		REST	EXER.	REST	EXER.	REST	EXER.	REST	EXER.	REST	EXER.
1 E. B.	II	22	39	98	95.5	218	542	6.25	8.26	3.61	4.78
2 B. B.	II	28									
4 H. Bo.	IV	34	56	93	86	218	271	3.91	3.30	2.09	1.76
5 P. C.	II	33	48	96	93						
6 E. C.	II	35	70								
7 S. D.	I	34	66	92	87						
8 B. F. (1)	II	25		96	97						
9 B. F. (2)	II	34	43	97	97	212	434	5.06	5.52	3.20	3.49
10 J. G.	I	17	38	95	93						
11 M. H.	III	23	48	86							
12 E. J.	I	30	40	97	90						
13 J. K.	I	25	41	95	97	235	579	5.5	9.3	3.11	5.25
14 E. K.	II	21	43	97	99						
15 A. L. (1)	III	31	43	93	93						
16 A. L. (2)	III	30	70								
17 O. L.	III	56	85	90	86						
18 H. M. (1)	I	28	39	97.5	95						
19 H. M. (2)	I	19	34	95	94	198	680	4.01	9.03	2.44	5.5
20 J. M.	III	28	31	96	95		661		7.7		4.05
21 A. M.	III	25	39	96	94	266	448	6.2	6.9	3.78	4.20
23 M. O.	II	22	34	96	95	189	685	4.76	8.73	2.59	4.75
24 L. R. (1)	II	35	84	94	85						
25 L. R. (2)	III	38	56	94	95	206	591	3.2	6.3	1.84	3.62
26 W. R.	I	33	42	97.5	95.5						
27 E. S.	I	25	39	97	95.5						
28 J. So.	II	33		98	98	318		3.6		1.73	
29 J. Sz.	IV	75	112	96	90						
30 H. T.	IV	35	52								
31 J. T.	II	48	65	97	95	198	410	3.5	4.7	2.2	2.96
32 W. W.	II	35	55								
33 P. W.	I	41	75	96.5	95	296	727	4.75	10.25	2.64	5.69

Class I.—

Considered as a group the ten studies performed on 9 patients an average of 6.5 years postoperatively showed the following average findings:

1. Total lung capacity, 3.97 L.
2. Vital capacity, 64.7 per cent of the normal predicted for a patient with two lungs.
3. Residual volume, 1.56 L.
4. Maximum breathing capacity, 58.1 per cent of the predicted value for a normal chest with both lungs.
5. First second vital capacity, 75.3 per cent.
6. Maximum midexpiratory flow, 1.57 L./sec.

7. Pulmonary artery systolic pressure: rest 28 mm. Hg.; exercise 46 mm. Hg.
8. Arterial oxygen saturation: rest 96 per cent; exercise 94 per cent.

Class II.—

The average findings of the group of 13 studies on 12 patients, an average 6.2 years postoperatively, were as follows:

1. Total lung capacity, 3.56 L.
2. Vital capacity, 57 per cent of the normal predicted for a patient with two lungs.
3. Residual volume, 1.47 L.

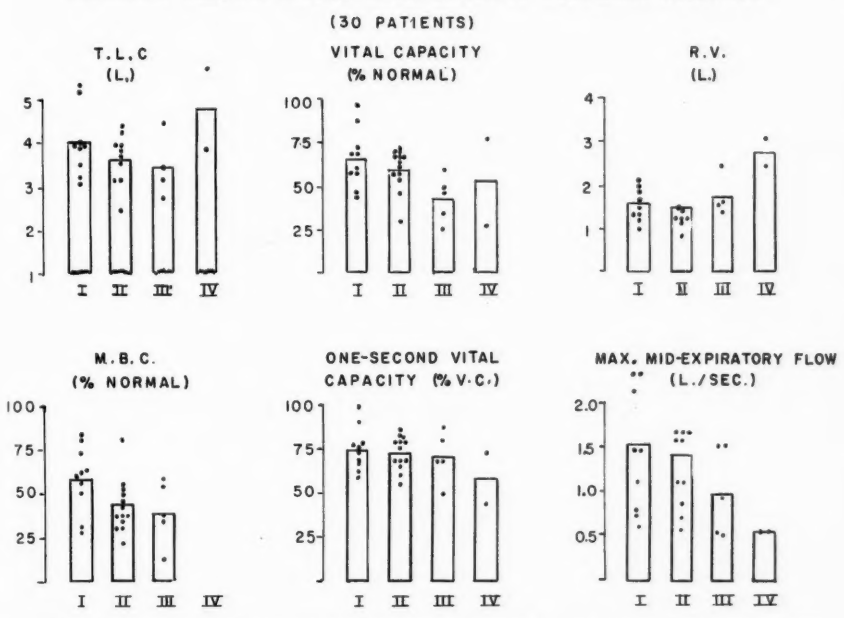


Fig. 1.—Results of pulmonary function studies grouped according to the patient's functional class. Bars represent average values for each group. Dots represent results of individual studies in each group. T.L.C. (L.) = total lung capacity in liters; Vital Capacity (% Normal) = per cent of vital capacity predicted for a normal subject with two lungs; R.V. (L.) = residual volume in liters; M.B.C. (% Normal) = per cent of the predicted maximum breathing capacity of a normal chest with both lungs; One-Second Vital Capacity (% V.C.) = per cent of the vital capacity expired in the first second; Max. Mid-Expiratory Flow (L./Sec.) = maximum midexpiratory flow in liters per second (16); I, II, III, IV = the functional class of the patients in each group. (See text for definition.)

4. Maximum breathing capacity, 44 per cent of the predicted value for a normal chest with both lungs.
5. First second vital capacity, 74 per cent.
6. Maximum midexpiratory flow, 1.44 L./sec.
7. Pulmonary artery systolic pressure: rest 31 mm. Hg; exercise 53 mm. Hg.

8. Arterial oxygen saturation: rest 96 per cent; exercise 94.5 per cent.

Class III.—

The average findings of the group of 7 studies on 6 patients, an average of 4.5 years postoperatively, were as follows:

1. Total lung capacity, 3.41 L.
2. Vital capacity, 42 per cent of the normal predicted for a patient with two lungs.
3. Residual volume, 1.75 L.
4. Maximum breathing capacity, 38.5 per cent of the predicted value for a normal chest with two lungs.
5. First second vital capacity, 72.4 per cent.
6. Maximum midexpiratory flow, 0.988 L./sec.

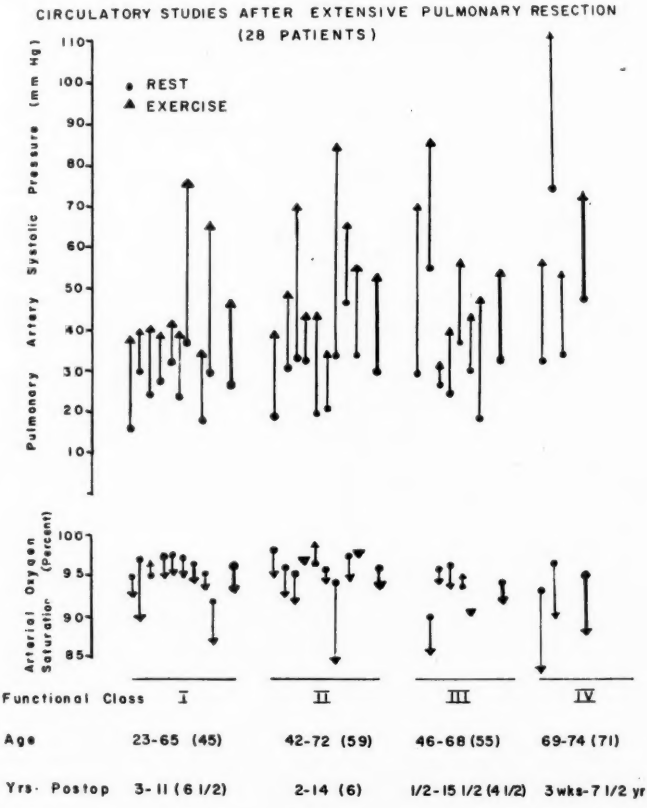


Fig. 2.—Pulmonary artery pressures and arterial oxygen saturations during rest and exercise, grouped according to the patient's functional class.

The lighter vertical lines represent the changes during exercise for each subject; the heavier vertical lines at the extreme right of each group represent the averages for the groups; Functional Class—(see text for definition); Age = the range of ages of patients within each group and the average age for the group; Yrs. Postop. = the range of the number of years after surgery for the patients in each group and the average for the group.

7. Pulmonary artery systolic pressure: rest 33 mm. Hg; exercise 53 mm. Hg.
8. Arterial oxygen saturation: rest 94 per cent; exercise 92 per cent.

Class IV.—

The average findings of the group of 3 patients studied an average of 5.25 years postoperatively were as follows:

1. Total lung capacity, 4.73 L.
2. Vital capacity, 53 per cent of the normal predicted for a patient with two lungs.

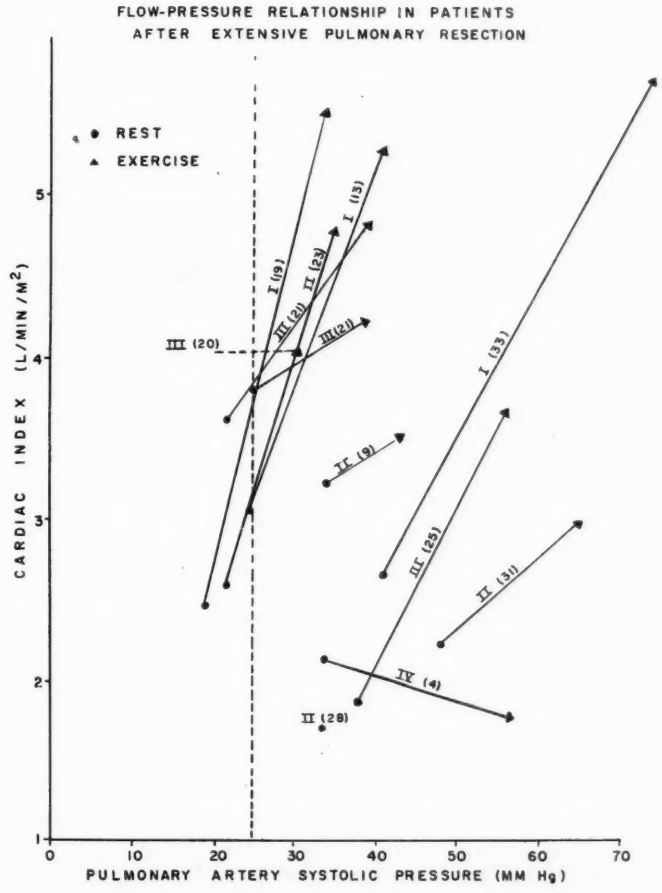


Fig. 3.—Relationship of pulmonary artery pressure to pulmonary blood flow during rest and exercise.

Each line represents the change during exercise for 1 patient; dots represent single determinations at rest or during exercise; numbers in parentheses identify patients by case number (see Table I); roman numerals signify the functional class of each case; vertical dotted line represents the pulmonary artery pressure level in normal subjects at rest and during exercise.

3. Residual volume, 2.71 L.
4. Maximum breathing capacity (obtained in only 1 case).
5. First second vital capacity, 60 per cent.
6. Maximum midexpiratory flow, 0.560 L./sec.
7. Pulmonary artery systolic pressure: rest 48 mm. Hg; exercise 73 mm. Hg.
8. Arterial oxygen saturation: rest 94.5 per cent; exercise 88 per cent.

A graphic representation of the relationship of the pulmonary blood flow to the pulmonary artery pressure in the 12 patients studied is presented in Fig. 3 and will be discussed later.

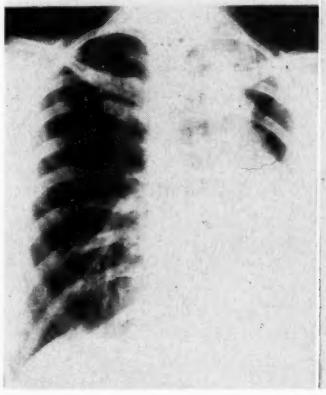
DISCUSSION

The results of the pulmonary function studies on this group of patients form the basis of a subsequent report and will not be considered fully here. However, in considering the relationship between the findings of the pulmonary function studies and the patients' functional capacities, as illustrated in Fig. 1, a number of generalizations were drawn. There appeared to be a correlation between the functional capacity and abnormalities in the maximum breathing capacity, residual volume, and maximum midexpiratory flow.[16] In the patients with increasing amounts of functional impairment, the maximum breathing capacity and maximum midexpiratory flow values became progressively more reduced, while, in these same patients, the residual volume became greater. This observation would support the impression that in these patients there were emphysematous changes in the remaining lung as represented by marked overdistention, increased airway resistance, as well as arterial oxygen unsaturation and abnormalities in the alveolar gas mixing (abnormal nitrogen washout curves). It should be pointed out that although a patient may have presented the picture of a markedly overdistended lung with marked herniation to the opposite side on x-ray studies, this did not necessarily represent emphysematous changes in that lung; other portions of the study revealed relatively normal findings and the patient experienced little or no functional disability. Patient 13 (J. K., Fig. 4) represents such a case.

Considering the pulmonary artery pressures with regard to the functional capacity of the patient, it was found that in Class I, 5 of the 8 patients displayed normal pulmonary artery pressures at rest but experienced increases from 25 to 120 per cent over resting levels during the exercise period. The remaining 3 patients in Class I had resting pulmonary artery pressures elevated 16 to 64 per cent above normal resting levels, and these levels during exercise increased 27 to 94 per cent above the resting levels. Five of the 12 patients in functional Class II had normal resting pulmonary artery pressure; during exercise these pressures rose 55 to 105 per cent above the resting levels. The 7 remaining patients in Class II presented right heart pressures elevated 32 to 52 per cent above normal resting values. During exercise, these pressures became further elevated to levels 35 to 140 per cent above the elevated resting levels. In the Class III group of cases, 3 of the 7 patients had normal resting pulmonary artery pressures that increased 18 to 110 per cent above this level during the

exercise period. The other 4 Class III patients at rest had pulmonary artery pressures elevated 16.5 to 125 per cent above normal. While exercising, these patients produced 39 to 133 per cent increases in pressure. All 3 of the Class IV patients displayed elevated resting right heart pressures; in 2 cases 40 per cent above the normal resting value and in the third case the elevation during rest was 200 per cent higher than normal. During the exercise period these pressures increased 48.5 to 65 per cent above the elevated resting values.

Pneumonectomy, as well as extensive bilateral pulmonary resection, results in a marked reduction of the total pulmonary capillary bed. Normally only a portion of the total capillary bed of the lung functions at one time. During exercise, with an increase in the amount of oxygen uptake in the lungs associated with increases in cardiac output, undoubtedly a greater fraction of the total lung capillary bed functions, as indicated by the lack of alteration in right heart pressure and arterial oxygen saturation in the normal individual.



FUNCTIONAL CLASS I.

T.L.C.	3.85 L.	
V.C.	2.05 L. (68%)	
R.V.	1.5 L.	
M.B.C.	57.0 L./min. (60%)	
1 sec V.C.	74%	
	Rest	Exer.
Pulm. Art. Press.	$\frac{25}{8}$ (14)	$\frac{41}{17}$ (25)
Art. O_2 Sat.	95%	97%
Pulse Rate	110.0	146.0
O_2 Consumption	235.0	579.0
Cardiac Output (L./min.)	5.5	9.3

Fig. 4.—A roentgenogram of Patient 13 (J. K.), a 37-year-old woman, taken 3 years post-pneumonectomy (left).

T.L.C. = total lung capacity; V.C. = vital capacity—per cent of normal predicted for subject with two lungs; R.V. = residual volume; M.B.C. = maximum breathing capacity—per cent of predicted value for a normal chest with both lungs; 1 sec. V.C. = per cent of the vital capacity expired in the first second; Pulm. Art. Press. = pulmonary artery pressures (mm. Hg) (mean pulmonary artery pressure in parentheses); Art. O_2 Sat. = arterial oxygen saturation; O_2 Consumption = oxygen consumption (ml./min.).

In the patient who has undergone pneumonectomy, the blood flow through the remaining lung is doubled. If the total capillary bed of the remaining lung does not change and the functioning capillary bed increases, then the reserve or nonfunctioning capillary bed of that lung is decreased. Consequently, when the oxygen requirements of the body are increased, such as by exercise, and the pulmonary blood flow increases, the reserve capillary bed may no longer be adequate to accommodate these demands. This would then result in an elevation of the right heart pressure and possibly a concomitant fall in arterial oxygen saturation.

The effect of the increased blood flow through the remaining capillary bed over the course of years is a matter for speculation and is under investigation. Possibly the increased flow leads to alterations in these vessels, which in turn

reduces the capillary bed and, in turn, the "reserve" capillary bed. However, the progressive reduction of the reserve capillary bed may be unrelated to the increase in blood flow and be related to pathologic processes occurring in the lung tissue with a secondary effect on the capillary bed, such as in emphysema. Undoubtedly, both or either process may play a role in an individual patient, as will be illustrated in later discussion.

It has been observed in normal subjects that the mean pulmonary arterial blood pressure did not change as the pulmonary blood flow increased up to 10 L./M.^{2} body surface area during a steady state of severe exercise.[17] Cournand[8] reported that in 5 subjects after pneumonectomy the mean pulmonary arterial pressure remained within normal limits with a pulmonary blood flow of less than 5 L./M.^{2} body surface area; the mean pulmonary arterial pressure rising progressively as the blood flow increased beyond this level.

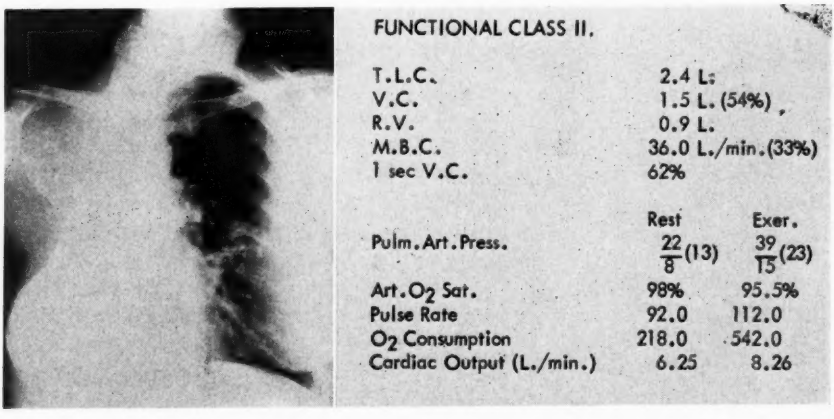

FUNCTIONAL CLASS II.
T.L.C. 2.4 L.
V.C. 1.5 L. (54%)
R.V. 0.9 L.
M.B.C. 36.0 L./min. (33%)
1 sec V.C. 62%
Rest Exer.
Pulm. Art. Press. 22/8 (13) 39/15 (23)
Art. O2 Sat. 98% 95.5%
Pulse Rate 92.0 112.0
O2 Consumption 218.0 542.0
Cardiac Output (L./min.) 6.25 8.26

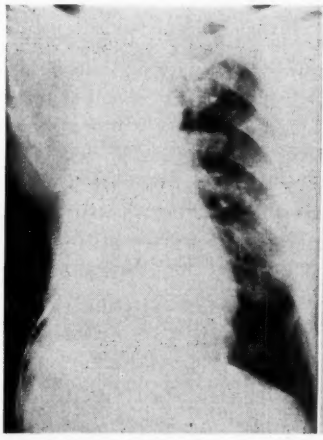
Fig. 5.—A roentgenogram of Patient 1 (E. B.), a 44-year-old woman, taken 8 years post-thoracoplasty (right) and 4 years postpneumonectomy (right). Same key as in Fig. 4.

In contrast to the findings of Cournand, the results were somewhat different in the group of patients in which the relationship of pulmonary blood flow to pulmonary artery pressure was determined (Fig. 3).

In 5 cases, No. 1 (E. B.; Fig. 5), No. 9 (B. F.), No. 21 (A. M.), No. 25 (L. R; Fig. 6), No. 31 (J. T.), despite the fact the pulmonary blood flow was less than 5 L./M.^{2} per minute, the pulmonary artery pressure increased. This suggested that the distensibility curve of the pulmonary vascular bed of these remaining lungs differed from that of normal lungs; i.e., a limitation in the expansibility of the vascular bed by a reduction in the number of small vessels, and/or a decrease in the cross-section of the lumen of the arterioles and precapillaries.

Three patients in our series, No. 17 (O. L.), No. 4 (H. Bo.), and No. 29 (J. Sz.), functional Classes III, IV, IV, respectively, demonstrated abnormalities in their pulmonary function studies indicating emphysema in the remaining lung. All of these patients had elevated resting pulmonary artery pressures

that increased markedly during mild to moderate exercise. One patient, No. 17 (O. L.), since the time of the study, has become a Class IV patient. Another patient, No. 4 (H. Bo.), has died since the study, presenting a clinical picture of progressive dyspnea and right heart failure in addition to metastatic disease.



FUNCTIONAL CLASS III.

T.L.C.	2.8 L.	
V.C.	1.2 L. (34%)	
R.V.	1.6 L.	
M.B.C.	31.0 L./min.(34%)	
1 sec V.C.	71%	
	Rest	Exer.
Pulm.Art.Press.	$\frac{38}{13}$(21)	$\frac{56}{30}$(39)
Art.O_2 Sat.	94%	95%
Pulse Rate	88.0	116.0
O_2 Consumption	206.0	591.0
Cardiac Output (L./min.)	3.2	6.3

Fig. 6.—A roentgenogram of Patient 25 (L. R.), a 63-year-old man, taken 15½ years post-pneumonectomy (right) and post-thoracoplasty (right). Same key as in Fig. 4.

FUNCTIONAL CLASS IV.

T.L.C.	5.66 L.	
V.C.	2.5 L. (75%)	
R.V.	3.03 L.	
M.B.C.	32.0 L./min.(43%)	
1 sec V.C.	46%	
	Rest	Exer.
Pulm.Art.Press.	$\frac{75}{35}$(48)	$\frac{112}{45}$(67)
Art. O_2 Sat.	96%	90%
Pulse Rate	84.0	92.0

Fig. 7.—A roentgenogram of Patient 29 (J. S.), a 74-year-old man, taken 7½ years post-pneumonectomy (left). Same key as in Fig. 4.

The third patient, No. 29 (J. Sz.; Fig. 7), was nearly a complete respiratory cripple, experiencing marked dyspnea at rest. All 3 of these patients following pneumonectomy resumed relatively normal activity and only later did they experience the onset of progressive dyspnea leading to functional incapacity. The question of whether the emphysema process existed in the remaining lung prior to surgery, or whether pneumonectomy resulting in overdistention of the

remaining lung brought about the emphysema, cannot be answered from this study. Unfortunately preoperative pulmonary function studies of these patients were not available.

Two of the Class I patients, No. 7 (S. D.) and No. 33 (P. W.), were found to have markedly elevated pulmonary artery pressures at rest and during exercise. Pulmonary function studies in these 2 cases failed to reveal any striking abnormalities. Patient No. 33 (P. W.) was found to have a pulmonary blood flow during exercise of 5.69 L./M.^{2} per minute which may explain much of the increased pressure during exercise, but fails to explain the elevated resting pulmonary artery pressure with a blood flow of 2.64 L./M.^{2} per minute. One may speculate on the future of Patients No. 7 (S. D.) and No. 33 (P. W.). Will the pulmonary hypertension produce changes in the pulmonary vessels resulting in even more marked elevations of pulmonary artery pressures, or will future pulmonary function studies reveal a pathologic process in the lung parenchyma with the pulmonary hypertension representing the first manifestation of such a process?

Patient No. 24, 25 (L. R.) represented one of the few instances in which repeat studies have been performed on the same person after first observing pulmonary hypertension. The first 10 to 11 years after his pneumonectomy, this patient carried on almost unrestricted activity. Progressive dyspnea developed and in the 1½-year interval between studies he moved from Class II to Class III in spite of no significant differences in the pulmonary function studies or cardiac catheterization findings. In retrospect it is possible that this patient should have been classified as Class III in the first study, but due to the fact that he had a stoic personality, was assigned to Class II.

Patient No. 20 (J.M.), a Class III patient, represented the opposite type of problem. On the basis of pulmonary function studies and cardiac catheterization findings, it might be predicted that he should be in Class I. However, this patient was extremely apprehensive and was reluctant to exert himself physically, 6 months after pneumonectomy. Since the time of his investigation he has shown remarkable improvement in his functional capacity.

In comparing the findings in the group of patients who had undergone bilateral resection with those of the postpneumonectomy patients, it is not possible to conclude whether the removal of a comparable volume of lung tissue bilaterally is better tolerated than a pneumonectomy. The bilateral resection group studied is too small and involves younger individuals than the majority of the postpneumonectomy patients. Nevertheless, the possibility exists that some of the adverse effects observed after pneumonectomy could be attributed to the deviation of mediastinal structures and thereby to the impairment of ventilatory and cardiocirculatory function. Mediastinal shift was absent in the group of patients following extensive bilateral resection.

SUMMARY AND CONCLUSION

1. Physiologic studies were performed on 29 patients 3 weeks to 15½ years following pneumonectomy or extensive bilateral pulmonary resection.

2. Results of the studies were compared with the functional capacity of each individual.

3. The older the patient at the time he underwent pneumonectomy, the more likely he was to experience a reduction of functional capacity later.

4. Reduction of functional capacity correlated well with reduction of maximum breathing capacity and maximum midexpiratory flow, and correlated inversely with the increase in residual volume.

5. Comparison of the functional capacity with pulmonary artery pressures at rest and during moderate exercise suggested a direct relationship. As the functional incapacity increased, the levels of pulmonary artery pressure at rest and during exercise were found more elevated.

6. Reduction of functional capacity appeared to be more closely related to the effects of pulmonary hypertension than alterations in arterial oxygen saturation.

7. Pulmonary artery pressure–pulmonary blood flow relationships in the remaining lung after pneumonectomy differed from that of a normal lung and suggested a limitation in the expansibility of the vascular bed.

8. Mechanisms of development of pulmonary hypertension as a late result of pneumonectomy are suggested.

REFERENCES

1. Longacre, J. J., Carter, B. N., and Quill, L. McG.: An Experimental Study of Some of the Physiologic Changes Following Total Pneumonectomy, J. THORACIC SURG. **6**: 237, 1937.
2. Carter, B. N., Longacre, J. J., and Quill, L. McG.: A Study of the Changes in Cardiorespiratory Physiology Following Total Pneumonectomy in Young Developing Animals, J. THORACIC SURG. **7**: 326, 1938.
3. Longacre, J. J., and Johansmann, R.: An Experimental Study of the Fate of the Remaining Lung Following Total Pneumonectomy, J. THORACIC SURG. **10**: 131, 1940.
4. Lester, C. W., Cournand, A., and Riley, R. L.: Pulmonary Function After Pneumonectomy in Children, J. THORACIC SURG. **11**: 529, 1942.
5. Cournand, A., Himmelstein, A., Riley, R. L., and Lester, C. W.: A Follow-up Study of the Cardiopulmonary Function in Four Young Individuals After Pnuemonectomy, J. THORACIC SURG. **16**: 30, 1947.
6. Peters, R. M., Roos, A., Black, H., Burford, T. H., and Graham, E. A.: Respiratory and Circulatory Studies After Pneumonectomy in Childhood, J. THORACIC SURG. **20**: 484, 1950.
7. Cournand, A., and Berry, F. B.: The Effect of Pneumonectomy Upon Cardiopulmonary Function in Adult Patients, Ann. Surg. **116**: 532, 1942.
8. Cournand, A., Riley, R. L., Himmelstein, A., and Austrian, R.: Pulmonary Circulation and Alveolar Ventilation—Perfusion Relationships After Pneumonectomy, J. THORACIC SURG. **19**: 80, 1950.
9. Birath, G., Crafoord, C., and Rudström, P.: Pulmonary Function After Pneumonectomy and Lobectomy, J. THORACIC SURG. **16**: 492, 1947.
10. Burnett, W. E., Long, J. H., Morris, C., Rosemond, G. P., and Wester, M. R.: The Effect of Pneumonectomy on Pulmonary Function, J. THORACIC SURG. **18**: 569, 1949.
11. Adams, W. E., Perkins, J. F., Jr., Flores, A., Chao, P., and Castellanos, M.: The Significance of Pulmonary Hypertension as a Cause of Death Following Pulmonary Resection, J. THORACIC SURG. **26**: 407, 1953.
12. Adams, W. E.: Problems in Pulmonary Resection for Primary Lung Tumor in the Aged, J. Am. Geriatrics Soc. **2**: 440, 1954.
13. Darling, R. C., Cournand, A., and Richards, D. W., Jr.: Studies on the Intrapulmonary Mixture of Gases. Section III. An Open Circuit Method for Measuring Residual Air, J. Clin. Invest. **19**: 609, 1940.

14. Perkins, J. F., Jr., Adams, W. E., and Livingstone, H. M.: The Conversion of Millikan and Wood Type Oximeters Into Direct Writing Recording Instruments for Use in Surgery in Studies of Pulmonary Function and in Teaching Pulmonary Physiology, J. Lab. & Clin. Med. **40**: 457, 1952.
15. Van Slyke, D. D., and Neill, J. M.: Determination of Gases in Blood and Other Solutions by Vacuum Extraction and Manometric Measurement, J. Biol. Chem. **61**: 523, 1924.
16. Levallen, E. C., and Fowler, W. S.: Maximal Midexpiratory Flow, Am. Rev. Tuberc. **72**: 783, 1955.
17. Riley, R. L., Himmelstein, A., Motley, H. L., Weiner, H. M., and Cournand, A.: Studies of Pulmonary Circulation at Rest and During Exercise in Normal Individuals and in Patients With Chronic Pulmonary Disease, Am. J. Physiol. **152**: 372, 1948.

(For Discussion, see page 379)

EFFECT OF SEVERE UNILATERAL HYPOXIA ON THE PARTITION OF PULMONARY BLOOD FLOW IN MAN

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WHEN a moderate degree of hypoxia is induced in either animal or human subjects, an elevation of the pulmonary arterial pressure frequently occurs. This pressor response appears to be caused by pulmonary vasoconstriction, since the left atrial pressure is not altered and since the cardiac output is only moderately increased.[1-9]

Such observations led von Euler and Liljestrand[2] to suggest that local hypoxia might play a role in controlling the distribution of the pulmonary blood flow. They reasoned that a local reduction in the alveolar oxygen tension would constrict the vessels in the affected area, thereby diverting a portion of the blood carried by these channels through vessels in better aerated parts of the lungs. Several investigators have tested this hypothesis by inducing unilateral hypoxia. Although it has been shown that this stimulus can change the partition of the pulmonary blood flow in animals,[10-16] there has been conflicting evidence that this response can be elicited in man.[17, 19]

In a previous series of experiments in this laboratory, the administration of 10 per cent oxygen to one lung in 6 human subjects had no observable effect on the partition of the pulmonary blood flow.[18] The present study was undertaken to investigate the effects of more severe hypoxia. In this study, 5 per cent oxygen was administered to one lung.

METHOD

The details of the method used to measure the blood flow through each lung have been described previously.[18] In brief, the technique combined right heart catheterization, bronchospirometry, and arterial cannulation. The simultaneous use of these three devices made it possible to measure (1) the gas exchange in each lung separately, (2) the concentration of oxygen in the arterial and mixed venous blood, and (3) the pressures in the pulmonary and brachial arteries.

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*Fellow of the Nuffield Foundation.

TABLE I. EFFECT OF UNILATERAL HYPOXIA ON GAS EXCHANGE AND BLOOD GAS COMPOSITION*

SUBJECT	STATE	$\dot{V}_{E_H}$ (L./MIN.)	$\dot{V}_{E_L}$ (L./MIN.)	$\dot{V}_{E_T}$ (L./MIN.)	$V_{O_{2_H}}$ (ML./MIN.)	$\dot{V}_{O_{2_L}}$ (ML./MIN.)	$\dot{V}_{O_{2_T}}$ (ML./MIN.)	$\dot{V}_{CO_{2_H}}$ (ML./MIN.)	$\dot{V}_{CO_{2_L}}$ (ML./MIN.)	$\dot{V}_{CO_{2_T}}$ (ML./MIN.)	R_{E_H}	R_{E_L}	R_{E_T}	CAP_{O_2} (ML./100 ML.)	Ca_{O_2} (ML./100 ML.)	(Ca_{O_2} minus $C\bar{v}_{O_2}$) (ML./100 ML.)
Group I. Patients With Minimal Unilateral Lung Disease																
B. M.	Control	4.65	5.12	9.77	123	100	223	75	81	156	.61	.81	.70	17.5	16.3	3.8
	Hypoxic	5.11	6.00	11.11	212	17	229	84	77	161	.40	4.5	.70	17.1	15.6	2.6
A. M.	Control	5.14	4.22	9.36	147	80	227	120	77	197	.82	.96	.87	14.2	13.1	3.5
	Hypoxic	5.62	4.72	10.34	231	–7	224	125	65	190	.54	–9.3	.85	14.2	12.0	3.8
D. H.	Control	5.66	5.54	11.20	171	82	253	121	100	221	.71	1.22	.87	17.8	16.8	3.1
	Hypoxic	7.30	6.34	13.64	294	1	295	174	82	256	.59	82.0	.87	17.8	16.5	3.1
	Control	6.51	5.74	12.25	210	76	286	144	96	240	.69	1.26	.84	17.8	16.6	3.2
S. L.	Control	5.71	5.03	10.74	153	104	257	95	81	176	.62	.78	.68	19.4	18.8	3.4
	Hypoxic	4.98	4.63	9.61	227	1	228	96	70	166	.42	70.0	.73	19.4	16.4	3.0
	Control	5.23	4.61	9.84	135	106	241	103	80	183	.76	.75	.76	19.4	17.9	3.1
J. Mc.	Control	5.21	6.44	11.65	129	166	295	95	128	223	.74	.77	.76	18.2	16.8	2.9
	Hypoxic	6.00	8.02	14.02	272	21	293	115	140	255	.42	6.7	.87	18.2	12.6	3.0
	Control	5.00	6.30	11.30	141	173	314	100	134	234	.71	.77	.75	18.2	16.3	3.6
Group II. Patients With Lobectomy and "Denervated" Lung																
P. H.	Control	4.15	4.75	8.90	159	117	276	94	102	196	.59	.87	.71	19.1	18.1	3.5
	Hypoxic	5.70	5.70	11.40	270	10	280	127	86	213	.47	8.6	.76	18.6	15.6	3.8
	Control	4.52	4.73	9.25	150	96	246	90	85	175	.60	.89	.71	18.5	16.9	4.0
R. J.	Control	5.80	5.36	11.16	154	120	274	133	120	253	.86	1.0	.92	20.0	19.1	5.1
	Hypoxic	4.66	4.72	9.38	248	13	261	121	81	202	.49	6.2	.77	20.1	17.0	4.1
	Control	5.50	5.38	10.88	181	117	298	122	111	233	.67	.95	.78	19.8	18.9	5.4

*The symbols used in Tables I, II, and III are defined as follows[23]: $\dot{V}_E$, volume of gas expired per minute (BTPS); $\dot{V}_{O_2}$, volume of oxygen taken up per minute (STPD); $\dot{V}_{CO_2}$, volume of carbon dioxide expired per minute (STPD); R_E, respiratory exchange ratio; CAP_{O_2}, arterial blood oxygen capacity; Ca_{O_2}, arterial blood oxygen content; $C\bar{v}_{O_2}$, mixed venous blood oxygen content; Sa_{O_2}, arterial blood oxyhemoglobin saturation; $\dot{Q}$, rate of volumetric blood flow.

The sub-subscripts H, L, and T refer to the high oxygen lung, the low oxygen lung, and both lungs, respectively.

The protocol consisted of three consecutive breathing periods. In the first, the lung free of disease breathed 25 per cent oxygen while the opposite lung breathed 21 per cent oxygen. Pressures were recorded at 3-minute intervals, and at the tenth and fifteenth minutes, samples of blood and gas were collected for the calculations of flow.

During the second period, unilateral hypoxia was induced by substituting 5 per cent oxygen for the 21 per cent oxygen mixture. Pressures were recorded every 3 minutes, and the arterial blood oxyhemoglobin saturation was measured every 4. The blood and gas samples used to measure flow were collected at the fifteenth and twenty-fifth minutes of hypoxia.

The third period was completed in five of the seven studies. During this period, 21 per cent oxygen was substituted for the 5 per cent oxygen mixture so that final control measurements could be obtained. Pressures were recorded at 3-minute intervals, and at the fifteenth minute the last collections of blood and gas were made.

CALCULATIONS

The total pulmonary blood flow was calculated by applying the Fick principle. This entailed dividing the total oxygen uptake by the arteriovenous oxygen difference. The flow through the lung breathing the high oxygen mixture was estimated by the same principle, but here it was assumed (1) that the saturation of the blood leaving this lung was 98 per cent, and (2) that its content of oxygen was equal to that calculated from the expression shown below.[18, 20]

$$0.98\ (O_2\ \text{Capacity} - 0.5) + 0.3.$$

Finally, the flow through the opposite lung was estimated by subtracting the flow through the high oxygen lung from the total pulmonary flow.

SUBJECTS

Two groups of subjects were studied. The first included 5 men believed to have normal pulmonary vessels: one (A. M.) had recovered from pneumonia; one (D. H.) had shown complete resolution of a lung abscess; and 3 had been admitted to the hospital for treatment of a minimal tuberculous focus at the apex of one lung.

The second group was composed of 2 men who had each undergone a modified right upper lobectomy for bronchogenic carcinoma approximately 14 months prior to the time of the study. In the course of the surgery, the proximal end of the divided right main-stem bronchus had been anastomosed to the distal end of the intermediate bronchus, and all of the adjacent tissues, including the nerves, had been divided. The pulmonary artery had also been stripped of adventitia and the adjacent nerves sectioned. Hence, only a nerve supply lying near the inferior pulmonary vein could have remained intact. It was believed, therefore, that the right middle and lower lobes were almost completely denervated.

RESULTS

The results are presented in Tables I, II, and III.

In Tables I and II, the initial control values represent an average of two successive measurements. In Table III, the partition of the ventilation and blood flow in the individual control measurements is shown. The measurements made after 15 minutes of unilateral hypoxia are not recorded in the tables since, in several instances, this period proved to be too brief for the attainment of a steady state of the gas exchange.

TABLE II. EFFECT OF UNILATERAL HYPOXIA ON ARTERIAL BLOOD OXYHEMOGLOBIN SATURATION, PARTITION OF TOTAL PULMONARY BLOOD FLOW, AND PULMONARY ARTERIAL PRESSURE

SUBJECT	STATE	$S_{a_{O_2}}$ (%)	$\dot{Q}_H$ (L./MIN.)	$\dot{Q}_L$ (L./MIN.)	$\dot{Q}_T$ (L./MIN.)	$\frac{\dot{Q}_L}{\dot{Q}_T} 100$ (%)	PULMONARY ARTERIAL PRESSURE (MM. HG) S.	D.	M.
Group I. Patients With Minimal Unilateral Lung Disease									
B. M.	Control	96	2.73	3.14	5.87	53	23*	4*	
	Hypoxic	92	5.89	2.92	8.81	33	23*	4*	
A. M.	Control	96	3.59	2.90	6.49	45	20	9	15
	Hypoxic	87	4.20	1.69	5.89	29	22	11	16
D. H.	Control	96	4.75	3.41	8.16	42			
	Hypoxic	94	7.54	1.98	9.52	21			
	Control	95	5.38	3.56	8.94	40			
S. L.	Control	99	4.50	3.06	7.56	41	16	8	11
	Hypoxic	86	4.20	3.40	7.60	45	16	8	11
	Control	94	3.38	4.39	7.77	56	16	8	11
J. Mc.	Control	95	3.49	6.68	10.17	66	25	11	15
	Hypoxic	70	3.40	6.37	9.77	65	37	14	22
	Control	91	2.88	5.84	8.72	67	29	13	17
Group II. Patients With Lobectomy and "Denervated" Lung									
P. H.	Control	97	4.18	3.71	7.89	47	27	10	16
	Hypoxic	85	4.35	3.02	7.37	41	35	14	20
	Control	93	3.00	3.15	6.15	51	25	9	15
R. J.	Control	97	2.85	2.52	5.37	47	28	16	21
	Hypoxic	86	3.76	2.61	6.37	41	34	19	25
	Control	97	3.17	2.35	5.52	43	28	16	21

*Pressures recorded from right ventricle.

In general, the values of ventilation and cardiac output measured during the control periods were larger than those usually found in resting subjects. These large values, which were also observed in the previous study,[18] were partly attributable to the presence of the bronchospirometry catheter, and partly to the dose of atropine given to suppress bronchial secretions. The values measured after 25 minutes of hypoxia were also large but satisfied the steady-state criteria.[5] In each of the subjects, both the total oxygen uptake and the respiratory quotient measured at the end of 25 minutes were comparable to those measured before hypoxia was induced.

In 3 of the subjects in the first group (A. M., D. H., and B. M.) unilateral hypoxia reduced the fraction of the total blood flow perfusing the hypoxic lung. The sequence of events in Subject A. M. is illustrated in Fig. 1. It

TABLE III. PARTITION OF VENTILATION AND BLOOD FLOW BETWEEN THE TWO LUNGS DURING THE CONTROL AND HYPOXIC PERIODS

SUBJECT	STATE	TIME OF EXPOSURE (MIN.)	$\frac{\dot{V}_{E_L}}{\dot{V}_{E_T}} \times 100$ (%)	$\frac{\dot{Q}_L}{\dot{Q}_T} \times 100$ (%)
Group I. Patients With Minimal Unilateral Lung Disease				
B. M.	Control	8	52	49
	Control	12	53	56
	Hypoxic	26	54	33
A. M.	Control	12	44	44
	Control	16	46	45
	Hypoxic	27	46	29
D. H.	Control	11	49	42
	Hypoxic	26	46	21
	Control	17	47	40
S. L.	Control	17	47	41
	Hypoxic	25	48	45
	Control	12	47	56
J. Mc.	Control	10	56	66
	Control	16	55	66
	Hypoxic	28	57	65
	Control	12	56	67
Group II. Patients With Lobectomy and "Denervated" Lung				
P. H.	Control	12	53	46
	Control	16	53	47
	Hypoxic	26	50	41
	Control	16	51	51
R. J.	Control	13	48	47
	Control	20	48	48
	Hypoxic	30	50	41
	Control	14	49	43

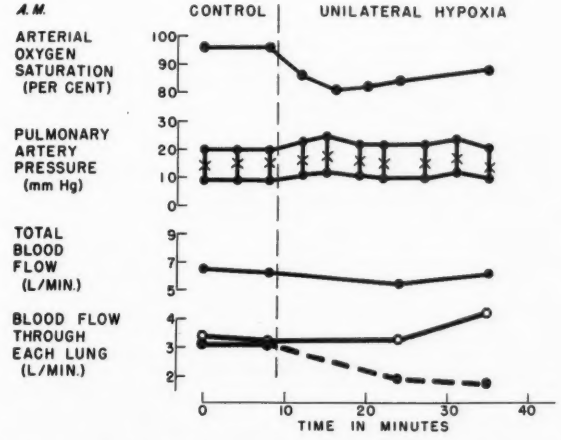

Fig. 1.—Effect of inducing severe unilateral hypoxia in Subject A. M. In the bottom graph, open circles and solid line indicate blood flow through the lung breathing the high oxygen mixture; closed circles and dashed line indicate blood flow through the hypoxic lung.

can be seen that the induction of hypoxia produced a prompt fall in the arterial oxyhemoglobin saturation. By the fifth minute of hypoxia, the saturation had reached its lowest level, after which it began to rise. At the end of 15 minutes, a change in the partition of flow was apparent, and at the end of the twenty-fifth minute, this change was quite pronounced. The sequence

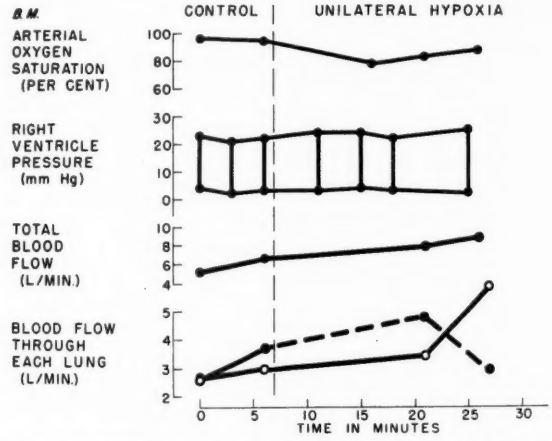

Fig. 2.—Effect of inducing severe unilateral hypoxia in Subject B. M. In the bottom graph, open circles and solid line indicate blood flow through the lung breathing the high oxygen mixture; closed circles and dashed line indicate blood flow through the hypoxic lung.

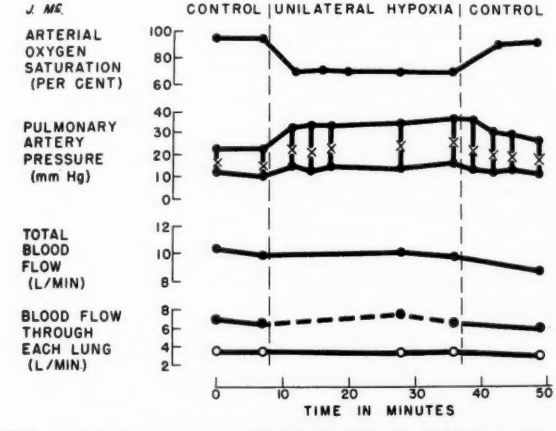

Fig. 3.—Effect of inducing severe unilateral hypoxia in Subject J. Mc. In the bottom graph, open circles and solid line indicate blood flow through the lung breathing the high oxygen mixture; closed circles and dashed line indicate blood flow through the hypoxic lung.

of events in Subject D. H. was similar, but that in Subject B. M. was somewhat different and is shown in Fig. 2. From the figure it can be seen that no shift in flow was evident at the fifteenth minute of hypoxia, but a shift appeared after 25 minutes had elapsed.

The results in Subject S. L. were inconclusive. After 25 minutes of hypoxia, the partition of flow was the same as it had been during the initial control period. However, the final control measurement indicated that the lung which had been hypoxic was carrying an increased fraction of the total pulmonary flow.

The response of Subject J. Mc. is illustrated in Fig. 3. Following the induction of unilateral hypoxia, the arterial oxyhemoglobin saturation fell to 70 per cent and remained at this low level. At the same time, pulmonary hypertension developed, but the partition of flow between the lungs remained identical to that during the control periods.

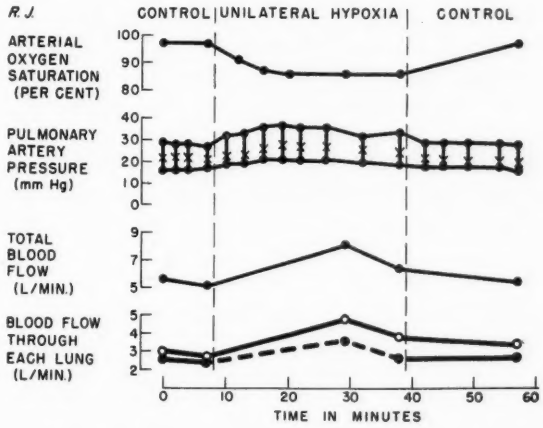

Fig. 4.—Effect of inducing severe uniateral hypoxia in Subject R. J. who had a denervated lung. In the bottom graph, open circles and solid line indicate blood flow through the lung breathing the high oxygen mixture; closed circles and dashed line indicate blood flow through the hypoxic lung.

In the 2 subjects in the second group, the denervated lung breathed the 5 per cent oxygen mixture. During hypoxia, the fraction of the total flow perfusing this lung was somewhat diminished, while the pressure in the pulmonary artery definitely rose (Fig. 4).

DISCUSSION

Four points merit emphasis: (A) the limitations of the method, (B) the demonstration that unilateral hypoxia may affect the partition of the pulmonary blood flow, (C) the degree of hypoxia necessary to produce an effect on the pulmonary vessels, and (D) the mechanism whereby the effect of hypoxia is mediated.

A. *The Limitations of the Method.*—The reliability of the calculated values of blood flow through the individual lungs rests directly on the validity of the assumption that the oxygen content of the blood leaving the high oxygen lung can be estimated. The reasons for believing that the assumption is valid have

been considered in two previous reports.[18, 20] In brief, these reasons are (1) that in each case the lung receiving the 25 per cent oxygen mixture was normal, (2) that on a previous day the administration of 25 per cent oxygen to both lungs had produced an arterial blood oxyhemoglobin saturation of at least 98 per cent, (3) that in the lung breathing the high oxygen mixture, the calculated $P_{A_{O_2}}$ was approximately 120 mm. Hg, and (4) that in the initial control period, with the bronchospirometry catheter in place, the arterial oxyhemoglobin saturation was never lower than 95 per cent. It would seem, therefore, that the expression used to calculate content would closely estimate the concentration of oxygen in the blood leaving the high oxygen lung.

A second limitation of the method stems from the fact that when the two lungs breathe gas mixtures containing different concentrations of nitrogen, a difference exists between the tension of nitrogen in the mixed venous blood and that in the alveolar air. This difference persists regardless of the length of time allowed for the attainment of a steady state. Thus, in the lung breathing the lower concentration of nitrogen, the volume of this gas expired exceeds that inspired, while in the opposite lung the reverse situation obtains. This inequality must necessarily affect the calculation of oxygen uptake. But theoretical considerations indicate that the effect of this phenomenon introduces an error of the order of only 1 per cent.

B. *The Demonstration That Unilateral Hypoxia May Affect the Partition of the Pulmonary Blood Flow.*—From Table III it is apparent that the fractions of flow perfusing the low oxygen lung showed close agreement in the two measurements made in the initial control period. Hence, the changes in this figure produced by unilateral hypoxia in 3 of the subjects suggest that a shift in flow away from the hypoxic lung actually occurred. In general, these changes were smaller and less constant than those observed by Blakemore, Carlens, and Björkman[19] in patients with lung disease. These investigators also used a technique involving bronchospirometry, cardiac catheterization, and arterial cannulation. However, their method of calculation of partition of blood flow involved (1) equilibration of the gas in the hypoxic lung with the mixed venous blood, and (2) estimation of the arterial blood oxygen tension when a high concentration of oxygen (80 per cent) was breathed. From the published data, it is not possible to determine whether a steady state of the respiration and circulation was attained.

In our own studies, it was of interest that although the partition of ventilation between the two lungs was not affected by unilateral hypoxia, the fraction of the total carbon dioxide eliminated by the hypoxic lung was reduced. Rahn and Bahnson[14] previously reported a difference in the alveolar carbon dioxide tensions in the two lungs of dogs subjected to unilateral hypoxia. They suggested that the difference might either be due to the Bohr effect, or to the fact that unilateral vasoconstriction altered the ventilation-perfusion relationships in the lungs.

Of particular interest was the fact that unilateral hypoxia produced a change in the partition of carbon dioxide excretion in the 2 patients with denervated lungs, even though in these patients it was questionable whether a shift in flow occurred.

C. *The Degree of Hypoxia Necessary to Produce an Effect on the Pulmonary Vessels.*—Several investigators have demonstrated that the inhalation of 12 per cent oxygen by both lungs almost always elicits a pressor response in the pulmonary artery. Presumably, this response is caused by constriction of the pulmonary vascular bed. One might expect, therefore, that the administration of 10 per cent oxygen to one lung would cause unilateral pulmonary vasoconstriction. The fact that it did not[18] may be attributable to the conditions under which gas is exchanged in the hypoxic lung.

The following considerations deal with this question. They are based on the fact that the alveolar oxygen tension can be roughly calculated by first dividing the alveolar carbon dioxide tension by the respiratory quotient, and then by subtracting this number from the tension of oxygen in the trachea. If we assume that the barometric pressure is 760 mm. Hg, then the tracheal oxygen tension when both lungs breathe 12 per cent oxygen is 86 mm. Hg. If we further assume an alveolar carbon dioxide tension of 40 mm. Hg and a respiratory quotient of 0.80, then the alveolar oxygen tension is approximately 36 mm. Hg in both lungs.

When 10 per cent oxygen is breathed, the tension in the trachea is 71 mm. Hg. When this mixture is breathed by a single lung, the lung continues to expire carbon dioxide although the rate of oxygen uptake substantially falls. As a consequence, the respiratory quotient has been found to be consistently greater than 1.0. If we assume a value of 1.3, which was the average of the values measured in the previous study,[18] and if we again assume an alveolar carbon dioxide tension of 40 mm. Hg, then the alveolar oxygen tension in the hypoxic lung is approximately 41 mm. Hg. In short, when 10 per cent oxygen is inspired unilaterally, the alveolar oxygen tension in the hypoxic lung is higher than it would be if 12 per cent oxygen were being breathed by both lungs.

Similar calculations made with the data in Table I indicate that the administration of 5 per cent oxygen to one lung reduces the oxygen tension to below 35 mm. Hg. Hence, a very low concentration of inspired oxygen is needed in order to produce an effect in one lung comparable with that produced when 12 per cent oxygen is breathed by both.

D. *The Mechanism Whereby the Effect of Hypoxia Is Mediated.*—In earlier studies in this laboratory, the inhalation of 12 per cent oxygen by both lungs elicited a pressor response in the pulmonary artery in each of 2 patients who had undergone sympathectomy.[8, 21] The first patient had had bilateral resection of the cervical and upper four dorsal sympathetic ganglia; the second had had all of the sympathetic ganglia removed. The results in these 2 patients confirm those made earlier in animals,[2] namely, that the sympathetic

ganglia are not essential for the mediation of the hypoxic effect. In the present series, the results obtained in the patients with a denervated lung, although not conclusive, seem to support the concept that the effects of local hypoxia are not mediated through the sympathetic nerves.

Beyond showing that nerves may not be important in the mediation of the response, our data shed no light on the exact mechanism involved. The observations made in J. Mc., the normal subject, who developed pulmonary hypertension without a shift in flow, suggest that two mechanisms may be present. On the one hand, the shift in flow may be caused by pulmonary hypoxia, as a result of a local action not yet clarified. On the other, the rise in the pulmonary arterial pressure would seem to indicate that both lungs were affected; a response which may be related somehow to the oxygen tension in the blood.[22]

SUMMARY

The administration of 5 per cent oxygen to one lung in 5 normal subjects reduced the fraction of the total flow perfusing the hypoxic lung in 3. In the fourth, the results were inconclusive, and, in the fifth pulmonary hypertension developed with no change in the partition of flow.

In 2 subjects in whom most of the nerves to one lung had been sectioned, unilateral hypoxia raised the pulmonary arterial pressure and produced a questionable shift in flow.

It appears that unilateral hypoxia can affect the partition of the blood flow between the lungs in human subjects. However, this effect is not invariably present and is seen only when the concentration of oxygen in the inspired gas mixture is very low.

REFERENCES

1. Beyne, J.: Influence de l'anoxémie sur la grande circulation et sur la circulation pulmonaire, Compt. Rend. Soc. de biol. **136:** 399, 1942.
2. Von Euler, U. S., and Liljestrand, G.: Observations on the Pulmonary Arterial Blood Pressure in the Cat, Acta physiol. scandinav. **12:** 301, 1946.
3. Motley, H. L., Cournand, A., Werkö, L., Himmelstein, A., and Dresdale, D.: The Influence of Short Periods of Induced Acute Anoxia Upon Pulmonary Artery Pressures in Man, Am. J. Physiol. **150:** 315, 1947.
4. Westcott, R. N., Fowler, N. O., Scott, R. C., Hauenstein, V. D., and McGuire, J.: Anoxia and Human Pulmonary Vascular Resistance, J. Clin. Invest. **30:** 957, 1951.
5. Fishman, A. P., McClement, J., Himmelstein, A., and Cournand, A.: Effects of Acute Anoxia on the Circulation and Respiration in Patients With Chronic Pulmonary Disease Studied During the "Steady State," J. Clin. Invest. **31:** 770, 1952.
6. Doyle, J. T., Wilson, J. S., and Warren, J. V.: The Pulmonary Vascular Response to Short-Term Hypoxia on Human Subjects, Circulation **5:** 263, 1952.
7. Stroud, R. C., and Rahn, H.: Effect of O_2 and CO_2 Tensions Upon the Resistance of Pulmonary Blood Vessels, Am. J. Physiol. **172:** 211, 1953.
8. Fishman, A. P., Himmelstein, A., Fritts, H. W., Jr., Lahoz, F., and Cournand, A.: Unpublished data. Presented at the Respiration Section, American Physiological Society, April, 1954.
9. Nahas, G. G., Visscher, M. B., Mather, G. W., Haddy, F. J., and Warner, H. R.: Influence of Hypoxia on the Pulmonary Circulation of Non-Narcotized Dogs, J. Appl. Physiol. **6:** 467, 1954.
10. Moore, R. L., and Cochran, H. W.: The Effects of Closed Pneumothorax, Partial Occlusion of One Primary Bronchus, Phrenicectomy, and the Respiration of Nitrogen by One Lung on Pulmonary Expansion and the Minute Volume of Blood Flowing Through the Lungs, J. THORACIC SURG. **2:** 468, 1932.

11. Dirken, M. N. J., and Heemstra, H.: Adaptation of Lung Circulation to Ventilation, Quart. J. Exper. Physiol. **34**: 213, 1948.
12. Atwell, R. J., Hickam, J. B., Pryor, W. W., and Page, E. B.: Reduction of Blood Flow Through the Hypoxic Lung, Am. J. Physiol. **166**: 37, 1951.
13. Peters, R. M., and Roos, A.: Effect of Unilateral Nitrogen Breathing Upon Pulmonary Blood Flow, Am. J. Physiol. **171**: 250, 1952.
14. Rahn, H., and Bahnson, H. T.: Effect of Unilateral Hypoxia on Gas Exchange and Calculated Pulmonary Blood Flow in Each Lung, J. Appl. Physiol. **6**: 105, 1953.
15. Venrath, H., Lechtenbörger, H., Valentin, H., and Bolt, W:. Das Verhalten von Atmung und Kreislauf bei- uni-und bilateraler Sauerstoffmangelatmung. Ein Beitrag zur Kompensation akuter Hypoxie durch Kreislaufumstellung, Ztschr. Kreislaufforsch. **44**: 544, 1955.
16. Borst, H. G., Whittenberger, J. L., Berglund, E., and McGregor, N.: Effects of Unilateral Hypoxia and Hypercapnia on Pulmonary Blood Flow Distribution in the Dog, Am. J. Physiol. **191**: 446, 1957.
17. Jacobaeus, H. C., and Bruce, T.: A Bronchospirometric Study on the Ability of the Human Lungs to Substitute for One Another. II, Acta med. scandinav. **105**: 211, 1940.
18. Fishman, A. P., Himmelstein, A., Fritts, H. W., Jr., and Cournand, A.: Blood Flow Through Each Lung in Man During Unilateral Hypoxia, J. Clin. Invest. **34**: 637, 1955.
19. Blakemore, W. S., Carlens, E., and Björkman, S.: The Effect of Unilateral Rebreathing of Low Oxygen Gas Mixtures Upon the Pulmonary Blood Flow in Man, Surg. Forum Clin. Congress, Am. Coll. Surgeons, Philadelphia, 1955, W. B. Saunders Company, vol. 5, p. 691.
20. Cournand, A., Fritts, H. W., Jr., Harris, P., and Himmelstein, A.: Preliminary Observations on the Effects in Man of Continuous Perfusion With Acetylcholine of One Branch of the Pulmonary Artery Upon the Homolateral Pulmonary Blood Flow, Trans. A. Am. Physicians **69**: 163, 1956.
21. Fritts, H. W., Jr., Harris, P., Clauss, R. H., Odell, J. E., and Cournand, A.: The Effect of Acetylcholine on the Human Pulmonary Circulation Under Normal and Hypoxic Conditions, J. Clin. Invest. **37**: 99, 1958.
22. Cournand, A.: The Mysterious Influence of Unilateral Pulmonary Hypoxia Upon the Circulation in Man, Acta cardiol. **5**: 429, 1955.
23. Standardization of Definitions and Symbols in Respiratory Physiology, Fed. Proc. **9**: 602, 1950.

Discussion

(Papers by Adams [page 352] and Himmelstein [page 369] and Their Associates)

DR. ANDRE COURNAND, New York, N. Y.—I am greatly honored to have been invited to participate in the program this morning. I have not come to the podium to present a paper but, indeed, to pay my tribute to the memory of Dr. Evarts Graham. My associate and close collaborator for more than 17 years, Dr. Himmelstein, will present our paper. This is due him since the data to be reported and discussed were secured to a great extent because of his skill and his thorough knowledge of the several techniques used in combination.

My first encounter with Dr. Graham dates back to 1937, on the occasion of the meeting of this Association held at Saranac Lake. On the program there were four papers dealing with the application of physiological tests to problems of thoracic surgery. If I recall correctly, our own presentation met with little enthusiasm, probably not so much because the subject was novel, but because of the accent of the speaker! During the meeting I had been impressed by the admirably controlled and self-possessed appearance of one of the members of the Association. When he came to tell me that he was much interested in the content of my paper, I felt somewhat less distressed by the apparent lack of enthusiasm of the audience. Several years later, Dr. Graham repeated this kindly gesture when, in collaboration with Dr. Frank Berry, I presented at one of our meetings the results of our studies in patients before and after pneumonectomy. His remarks can be found in the discussion published in the Journal of Thoracic Surgery.

On a third occasion, Dr. Graham gave me an even more objective and precious evidence of his sympathy. In a way this was probably an opportunity for him to express his feeling toward Frenchmen whom he had learned to like during the First World War. I

had written him a letter of recommendation on behalf of Dr. Andre Germain, a Fellow of the French Cultural Service, who was sent to the United States to be trained as a thoracic surgeon. Within a week not only had I received a favorable answer but my protégé was starting to work as an assistant resident on the world-renowned surgical service at Barnes Hospital. This was a great privilege and opportunity for him. For me, the exchange of letters, and the rapid and favorable decision, was proof that Dr. Graham, like other great men, was not bound by precedents or by conformity to strict rules.

DR. PAUL NEMIR, JR., Philadelphia, Pa.—I wish to congratulate Dr. Adams and his associates on this excellent presentation. Studies such as these are important and will, I think, contribute equally as much in our efforts to improve long-term survival statistics as will the significant studies relating to blood vessel and lymph node invasion, and the studies reported by Dr. Meade.

Dr. Meade stated that in 32 per cent of the autopsies studied there was no evidence of recurrence. Undoubtedly a large number of the failures to survive for appreciable periods of time was due to the fact that pulmonary insufficiency occurred following the resectional therapy. This has certainly been true in an analysis of our own series of cases.

If it were possible by more accurate preoperative pulmonary function studies to delineate and exclude this group of patients, then we could expect better survival rates following resection.

While it is true that withholding operation from the patient with precarious pulmonary function is not going to prolong his survival, at least he will be spared the distressing situation of being a pulmonary cripple.

We have continued our studies of pulmonary function, utilizing the method of unilateral pulmonary artery occlusion reported before the Association 2 years ago. Our findings are in agreement with those reported by Dr. Adams. An elevated resting pulmonary artery pressure or a sustained increment following balloon occlusion of the unilateral pulmonary artery has been almost uniformly of grave prognostic significance.

We have found that small increments of the order of 6 to 8 mm. Hg following occlusion are of great significance. Since the average age of the patients we have studied has been 61 years, the control resting pulmonary artery pressures have been at the very upper limits of normal. Small increments could therefore be reasonably expected to have greater significance. I would like to ask Dr. Adams the magnitude of the elevations they have noted.

A second measurement which we have found to be important is the wedge pressure. Elevations of this pressure may also be correlated with a poor prognosis.

While it is true that these detailed studies may not be routinely performed, the knowledge we are gaining from them will allow for more accurate preoperative evaluation of the patient undergoing pulmonary resection, and I am sure that more simple methods for obtaining similar information will soon be available.

DR. WILLIAM S. BLAKEMORE, Philadelphia, Pa.—It is a fitting tribute to Dr. Graham to have a presentation of physiologic studies in this morning's program. One of Dr. Graham's earliest contributions was in the field of pulmonary physiology. The work which Dr. Himmelstein has presented is especially significant in corroborating the findings in man that were previously shown in animals. Several years ago Dr. Björkman, Dr. Carlens, and I performed similar studies in man, with one lung rebreathing a mixture of about 6 to 7 per cent oxygen and 5 per cent carbon dioxide and the other lung rebreathing a mixture of 50 to 80 per cent oxygen and less than 0.3 per cent carbon dioxide. We found that differences in oxygen and/or carbon dioxide concentrations in the inspired gas had an effect upon pulmonary blood flow. Our method differed from the method used by these investigators and we were disappointed that Fishman and this group did not find a change using 10 per cent oxygen in the hypoxic lung, since the method used in

these studies should provide greater accuracy. Now, however, they have today reported changes in blood flow with lower than 10 per cent oxygen in the hypoxic lung. Since even these methods of calculating the blood flow through each lung are likely to have a high per cent of error, additional studies using more direct methods, i.e., Forster's acetylene method for measuring the pulmonary blood flow, should be tried to re-evaluate the effects of different concentrations of carbon dioxide and oxygen; to determine the critical concentrations of these substances necessary to produce changes in distribution of the pulmonary blood flow; and to determine the receptors and mechanism that mediates these changes in the lung.

DR. ADAMS (Closing).—I wish to thank the discussers for their constructive contributions to our paper. The problem of reduced pulmonary reserve, especially with the development of pulmonary hypertension, continues to increase in frequency with the increase in life expectancy. Although much has been learned about the nature of this problem, methods of management are still far from adequate. Although I have been asked to present this paper, the major portion of the investigative work has been carried out by other authors on the paper.

SURGICAL TREATMENT OF "ATYPICAL" PATENT DUCTUS ARTERIOSUS

William P. Young, M.D. (by invitation), George G. Rowe, M.D. (by invitation), Anthony R. Curreri, M.D., and Joseph W. Gale, M.D., Madison, Wis.

THE USE of cardiac catheterization has frequently established the presence of patent ductus arteriosus in the absence of the usual physical signs. These patent ductus are usually associated with pulmonary hypertension. The term "atypical" patent ductus arteriosus has come to imply the presence of associated pulmonary hypertension. The advisability of closing a patent ductus when the pulmonary artery pressure approaches or equals the pressure in the aorta has been questioned on the basis of the high operative mortality.[1, 3, 4, 14] A number of successful results, however, have been reported by these and other authors.[6, 12, 13, 16, 17]

On the other hand, closure of the uncomplicated patent ductus is safe and unquestionably indicated.[8] At the University Hospitals, patients with the typical finding of patent ductus arteriosus are corrected without cardiac catheterization or other special diagnostic procedures. In 163 uncomplicated cases in which there was ligation or division, there were 2 deaths and 2 recurrences. There were no other significant complications. Among the early cases in 1940 and 1941, there were 4 ligations carried out in the presence of subacute bacterial endarteritis. Two of these patients died and account for the deaths in the series of typical cases. One of the recurrences followed ligation with umbilical tape, and it is felt that the tie was not drawn completely tight. The second recurrence followed closure of the ductus with nonabsorbable ties and suture ligatures without dividing the ductus. It is now routine to divide all patent ductus.

It is the purpose of this paper to report surgical experience with a group of 28 patients who had patent ductus with varying degrees of pulmonary hypertension. These patients were considered preoperatively to have a patent ductus arteriosus as the only anatomic lesion. Later, 3 were shown to have concomitant congenital heart defects, and a fourth patient is suspected of having an additional defect. The accompanying table lists the preoperative

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catheterization pressure in the pulmonary artery and aorta, pressures in the pulmonary artery and aorta taken at the time of surgery before and during temporary occlusion of the ductus, postoperative catheterization data, and available follow-up data. All but 1 patient had preoperative cardiac catheterization studies. The exception was a 3-month-old baby in whom a large patent ductus arteriosus was demonstrated by angiocardiogram. The cardiac catheter was passed through the ductus into the aorta in all but 2 patients, and in those 2 patients the systemic pressure was recorded from the femoral artery. Technical difficulties made some of the pressures recorded at the time of surgery unsatisfactory. For 5 of the cases, simplified pressure measurements without accurate calibration were carried out which showed only relative pressure changes. In each of these instances the systolic pressure increased in the aorta and decreased in the pulmonary artery with temporary occlusion of the patent ductus arteriosus. Postoperative catheterization pressures are given for 8 patients, the catheterization having been carried out from 7 to 14 months after surgery.

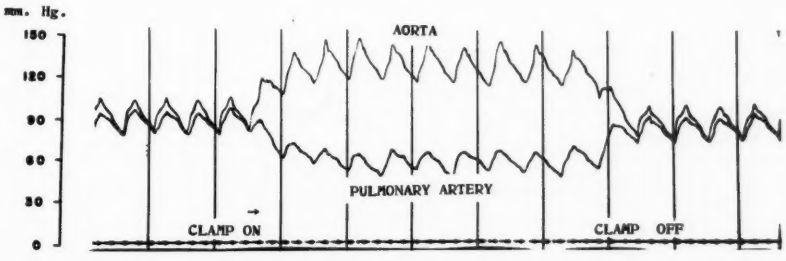

Fig. 1.—Optical recording of pressures in the aorta and pulmonary artery at surgery before, during, and after temporary occlusion of the patent ductus arteriosus. (Case 26, 12-year-old girl who had had intermittent bidirectional shunt through the ductus.) This response to temporary occlusion is very favorable. It established that the bidirectional shunt was predominantly left-to-right and also that there was a significant shunt so that the patient's hemodynamics will be improved by dividing the ductus.

The response in the pressures in the pulmonary artery and aorta during temporary occlusion at time of surgery appears to be very significant as far as the patient's prognosis is concerned. Fig. 1 shows a very favorable response to temporary occlusion. These are pressure tracings recorded in a 12-year-old girl who had had intermittent bidirectional shunt through the ductus. This response to temporary occlusion confirmed the catheterization data which indicated that the bidirectional shunt was predominantly left-to-right. It also established that the shunt was of significant volume so that the patient's hemodynamics could be improved by dividing the ductus. Varying the concentration of oxygen in the anesthetic mixture did not affect these pressures but probably would have if observation had been continued over a long period.[2]

Fig. 2 shows pressures similarly recorded in Case 5 who is now suspected of having an interventricular septal defect. Such a lack of response to occlusion of the ductus is considered an unfavorable sign in the presence

TABLE I

CASE NO.	AGE	SEX	PREOP. CATHETER. PRESSURES		PRESSURES TAKEN AT SURGERY				POSTOP. CATHETER. DATA			LAST REPORT (MONTHS POSTOP.)	FOLLOW UP		
					DUCTUS OPEN		DUCTUS CLOSED								
			PA	AORTA	PA	AORTA	PA	AORTA	MO. POSTOP.	PA	SYSTEMIC A		SYMPTOMS	ACTIVITY	FINDINGS
1	21	M	64/42	180/70 FA	68/38	106/62	41/23	132/103	14	29/9	140/86 FA	14	No	Full	Neg.
2	2	F	77/48	94/42	47/35	67/56	41/32	80/75				26	Alive and well (letter)		
3	9/12	F	65/39	103/61								6	No	Full	G 1 Sys. M§
4	11	F	90/57	114/60	100/80	115/78	60/47	170/115				36	Alive and well (letter)		
5	7/12	M	48/26	85/42 FA	63/32	82/51	63/32	84/50				2	No	Full	Systolic murmur; suspect VSD
6	4	F	77/40	77/40	98/93	105/90	84/78	129/114	12	66/31	82/51 FA	30	No	Full	G 1 Sys. M
7	18	F	48/34	97/50	40/28	88/53	26/9	133/80				19	No	Full	Neg.
8	2/12	F	78/60	68/50	50/43	48/43	52/43	50/47				Died 41st postop. day	Chronic passive congestion and diffuse necrosis of liver		
9	2	F	52/31	124/60	*							25	No	Full	Neg.
10	11	M	58/37	94/51	45/20	90/50	30/10	100/80				1	Recovering rapidly		G 1 Sys. M
11	9/12	M	58/25	68/23	80/55	80/47	61/27	106/50	7	37/10	64/54 FA	7	No	Full	Neg.
12	1	F	70/48	83/50	67/52	85/60	52/35	120/84				1	Recovering rapidly		
13	1	F	100/40	100/50	36/30	55/42	29/25	65/47				Died 18th postop. day of pneumonia			
14	1	F	85/50	80/60		80/50		100/70				7	No	Full	G 1 Sys. M
15	9/12	F	48/20	95/63								8	No	Full	Neg.
16	2	F	86/58	86/54	40/30	71/58	29/20	93/80				33	No	Full	Neg.
17	1	M	50/25	113/73								13	No	Full	Neg.
18	5	M	48/36	131/75	*							59	Alive and well (letter)		
19	29	M	86/48	127/64	*				12	32/18	118/77 BA	12	No	Full	Huge heart†
20	7/12	F	42/35	57/41	51/33	61/46	50/34	61/46				Died 2nd postop. day			ASD and VSD
21	10	F	101/63	112/60	71/56	76/62	56/37	90/77	8	80/36	125/75 BA	23	No	Full	G 1 Sys. M
22	3/12	M	Large PDA demonstrated on angiocardiogram		44/23	45/21	44/23	45/31				Died 1st postop. day			VSD
23	9/12	M	90/52	97/60	90/54	94/60	72/49	111/70				45	No	Full	G 1 Sys. M
24	11	F	103/70	109/70	84/74	93/77	67/57	117/102	13	103/53	126/75 FA	13	No	Full	‡
25	6	M	47/24	105/60					13	25/11	93/66 FA	25	No	Full	G 1 Sys. M
26	12	F	100/63	107/62	95/75	108/75	65/41	145/115				5	No	Full	G 1 Sys. M
27	4	F	74/58	94/56	*							14	No	Full	G 1 Sys. M
28	2	M	77/44	103/44	*				11	19/7	117/65 FA	11	No	Full	Neg.

*Simplified pressure measurements without accurate calibration showing only relative pressure changes—in each instance systolic pressure increased in the aorta and decreased in the pulmonary artery with temporary occlusion of the PDA.

†Patient died 23 months postoperative after a fight. Autopsy showed enlarged heart, no defects.

‡Intermittent Grade 1 systolic murmur, RV apex beat, ECG—right ventricular hypertrophy, suspected of having another defect, was recatheterized and none was found but has severe pulmonary hypertension.

§G 1 Sys. M. = Grade 1 systolic murmur.

of a ductus of significant size. The lack of response indicates the shunt through the ductus is not supporting the pulmonary hypertension and that the elevation of pulmonary artery pressure is due to an increase in pulmonary vascular resistance or another more significant shunt. When there was no pressure response to closure of a patent ductus, the prognosis in this series was poor. Three of 4 such patients died in the postoperative period. There was only one death among the other 24 patients who had a favorable pressure change with temporary occlusion of the ductus. The pressure responses do not need to be as marked as shown in Fig. 1 to be a favorable prognostic sign.

Another cardiovascular defect was found later in 3 of 4 patients who had no pressure change with temporary occlusion in the ductus. Four other patients not included in this series had patent ductus arteriosus and known septal defects. Systemic and pulmonary artery pressures were recorded while the ductus were being closed and no significant change occurred in the pressures. Considerable change, however, did take place with the closure of the septal defects. Adding these cases to the preceding group gives a total of 8 cases in which there was no pressure response to occlusion of the ductus.

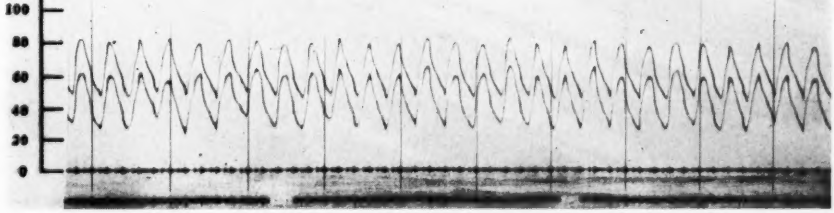

Fig. 2.—Optical recordings of pressures in the aorta and pulmonary artery at surgery before, during, and after temporary occlusion of the patent ductus arteriosus. Occlusion was established and released at the first and second breaks in the heavy line, respectively. (Case 5, 7-month-old boy.) This lack of response to occlusion is an unfavorable sign and indicates that the shunt through the ductus is not supporting the pulmonary hypertension. In this case it is suspected that there is a ventricular septal defect.

In 7 of these 8 instances, there were additional cardiac defects. The lack of pressure changes on temporary occlusion of the ductus may also be due to a balance between the systemic and pulmonary resistances but at least on theoretical grounds should be found only when the preocclusion pressures are equal in the pulmonary artery and aorta.

Many authors have described the changes in the pulmonary arteries. Edwards,[5] Smith,[14] and Cosh[3] have good discussions on the subject and it will not be discussed here except to say that in the presence of pulmonary hypertension the walls of muscular arteries are frequently found to be thickened with encroachment upon the lumen. There is reason to believe that in some instances there is persistence of the fetal pattern of blood vessels and in other instances the changes in the vessels are acquired as a result of a long-standing pulmonary hypertension.

...ventricular hypertrophy, suspected of having another defect, was recatheterized and none was found but has severe pulmonary hypertension
...Grade 1 systolic murmur, RV apex beat, ECG—right ventricular
§§ 1 Sys. M. = Grade 1 systolic murmur.

TECHNIQUE OF CLOSURE

In the technique of closing a patent ductus in the presence of pulmonary hypertension, one must be very conscious of the greater hazard of serious bleeding from a tear in the pulmonary artery. It is considered essential that the short wide ductus that have associated pulmonary hypertension be divided. Fig. 3 illustrates the technique that was used in closing many of these ductus. The aorta is dissected out above and below the ductus and umbilical tapes are passed about the aorta at these sites. The aorta is completely mobilized in the vicinity of the ductus and mobilization of the ductus is completed by dissecting medial to the aorta and ductus as suggested by Jones.[9] A moderate degree of hypothermia has been a routine for surgery for patent ductus since it was suggested by Ellis and his associates.[6] The patients were cooled to 92° to 95° F. These two procedures provide the opportunity to cross-clamp the aorta if necessary. The Potts-Smith clamp[9] is frequently used when there

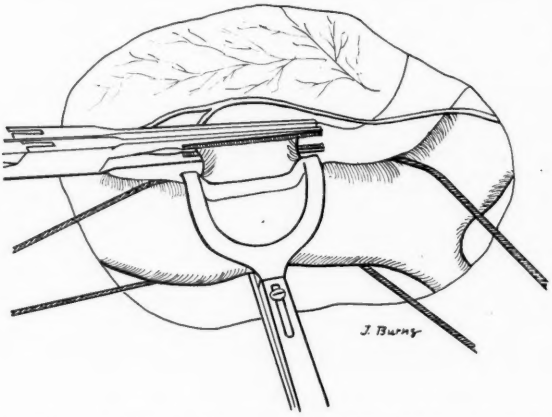

Fig. 3.—The aorta is mobilized adjacent to the ductus and tapes are passed around the aorta above and below the ductus. The vagus and recurrent nerves are carried toward the pericardium with the dissection. A Potts-Smith aortic clamp is frequently used so that the ductus may be divided flush with the aorta, permitting suturing of the ductus rather than the pulmonary artery in the presence of pulmonary hypertension.

is pulmonary hypertension or the ductus is short and wide. In such a situation the ductus itself is allowed to remain with the pulmonary artery to facilitate its closure—the ductus being divided flush with the aorta. A tear in the pulmonary artery is more difficult to control than a defect in the wall of the aorta when the aorta is mobilized. Controlled hypotension[7] with the use of trimethaphan camphorsulfonate (Arfonad) or the cross-clamping of the aorta[6] proximal to the ductus has been used to facilitate the application of clamps. Although the longitudinal suture line in the aorta, made necessary by the use of the Potts-Smith clamp, may be less desirable than the transverse suture line, there has been no trouble with healing of the longitudinal line and no patients have been lost due to technical difficulties.

Enlargement of the heart, pulmonary artery, and aorta may reduce the operating space about the ductus and require the employment of many of

the possible safe-guards, such as hypothermia, cross-clamping the aorta, controlled hypotension, Potts-Smith clamps, placing mattress sutures before cutting through the ductus, and dividing and suturing the ductus in stages.

The data in Case 10, shown in Table I, are of unusual interest. This patient, whose proved patent ductus had been divided 7 years earlier, presented himself again and on cardiac catheterization was found to have an aorticopulmonary window with pulmonary hypertension. This window is included with the atypical patent ductus because of the identical hemodynamics. With a pump-oxygenator ready, a bilateral anterior thoracotomy was made. The site of the previous ductus division was dissected out

Fig. 4.

Fig. 5.

Fig. 4.—An aorticopulmonary window is present adjacent to the ventricles. The site of the window can be seen just above the proximal tape around the aorta. The origin of the pulmonary artery may be seen as the ventricular musculature is retracted with the instrument (Case 10). There was moderate pulmonary hypertension.

Fig. 5.—Aorticopulmonary window has been divided and sutured. Controlled hypotension was maintained with Arfonad during the application of clamps and the suturing. A pump-oxygenator had been prepared but was not used.

and no recurrence found. Fig. 4 shows the aorticopulmonary window which measured 20 mm. in diameter and was just far enough above the ventricles so that it could be divided and sutured as one divides a ductus. Controlled hypotension was used to facilitate the application of clamps and suturing. The pump-oxygenator was not used. Fig. 5 shows the suture defects in the aorta and pulmonary artery after the communication had been divided.

RESULTS

If the data on Table I is summarized, it is found that 24 of the 28 patients had favorable pressure changes to temporary occlusion of the ductus. When

no pressure changes could be recorded, care was taken to observe the systemic pressure obtained by the anesthesiologist. Fortunately, in each of these instances there was a significant rise in systolic and diastolic pressure with temporary occlusion of the ductus. All but 1 of these 24 patients survived. The 1 patient (Case 13) died on the eighteenth postoperative day and postmortem examination showed pneumonia. There had been considerable pulmonary congestion in spite of the favorable pressure response. Four patients (Cases 5, 8, 20, and 22) did not have favorable pressure changes. Three of these died and 2 of those who died were found to have other cardiac defects. The third died with chronic passive congestion and diffuse necrosis of the liver and it is believed that the right heart failed due to a high pulmonary resistance. Case 24 is of interest because 13 months after division of the ductus following a favorable pressure change on temporary occlusion of the ductus she was found to have a variable systolic murmur and a right ventricular apex beat. Her electrocardiogram showed right ventricular hypertrophy. She was suspected of having another cardiac defect but on recatheterization no abnormality was found other than severe pulmonary hypertension. It is interesting that she remains asymptomatic. In none of these cases was a rise in the pulmonary pressure with temporary occlusion of the ductus encountered. Had such a change been found the ductus probably would not have been closed.

DISCUSSION

When the pulmonary artery and aortic pressures did not change on temporary occlusion of the patent ductus arteriosus, the patients probably did not survive because they were subjected to considerable surgical trauma without benefit to their hemodynamics. In 1 of the cases it is felt that there was a balance between the systemic and pulmonary resistance but in the other 3 cases it is felt there was no change because a more significant defect was not corrected. Failure of the pressures to change is a strong indication to search for other defects and correct them.

The test of temporary occlusion would be useful in separating the bidirectional shunt that has predominant left-to-right shunt from that which has predominant right-to-left shunt. Closure of the ductus in the presence of the predominant left-to-right shunt will usually be well tolerated while closure of the ductus in the latter situation may not be tolerated. The ductus is not a safety valve until a shunt is predominantly right-to-left.

The unusual patient with a right-to-left shunt of a degree such as to contraindicate closure of a patent ductus arteriosus should be able to be excluded from the group being taken to surgery by a clinical examination and cardiac catheterization. Fig. 6 shows a roentgenogram taken during cardiac catheterization of such a patient. A calcification of the aorta, pulmonary artery, and ductus arteriosus can be seen. The pulmonary artery pressure exceeded that in the aorta and the patient was grossly cyanotic. This catheterization was carried out in 1953 and it is interesting that she

remains essentially unchanged during the 5-year period since then. She continues to do limited housework. It seems likely that, when a patient reaches this stage in the syndrome, the more significant disease is in the pulmonary vessels rather than the ductus. Slowly occluding the ductus with fibrosing agents, partial closure of the ductus,[15] creating a smaller artificial ductus prior to closing the large ductus,[10] or creating a shunt into the right atrium or the distal divided end of the subclavian artery[12] are some of the suggestions that have been made to facilitate closure of patent ductus with reversed flow.

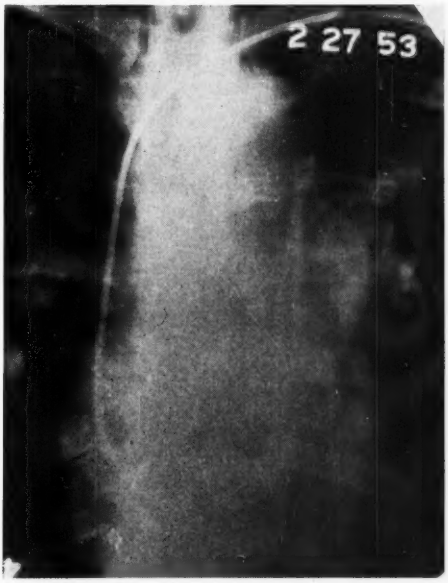

Fig. 6.—Roentgenogram taken at time of cardiac catheterization with the catheter through the ductus and down the aorta. Calcification can be seen in the pulmonary artery, ductus, and aorta (35-year-old woman with marked cyanosis, was not subjected to surgery). PA pressure 144/69 m94; aorta pressure 112/75 m88; O_2 saturation was 87 per cent. Right-to-left shunt was 1.8 L./min.; left-to-right shunt was 1.4 L./min.

None of these seem to attack the basic problem of the abnormal pulmonary vessels. Phlebotomy and controlled blood loss suggested by Sirak and Humphreys[13] may be helpful in preparing such patients for surgery as well as carrying them through the surgery. Lukas, Araujo, and Steinberg[11] have pointed out that polycythemia increases the right-to-left shunt because the increased viscosity of the blood produces an increased resistance to flow through the pulmonary vessels. They point out further that the polycythemia favors thrombosis in the sclerotic pulmonary arteries, further decreasing their capacity. It is rational to control the polycythemia and with this controlled, a few more of the patients might tolerate and be benefited by surgical closure of the ductus in the presence of considerable right-to-left shunt.

CONCLUSIONS

The diagnosis of patent ductus arteriosus in the presence of pulmonary hypertension is more difficult than in the absence of pulmonary hypertension. Cardiac catheterization is probably necessary to make the diagnosis and even then the demonstration of the patent ductus arteriosus may not establish that the ductus is the significant lesion.

While uncomplicated patent ductus may be closed with almost certain good results, the closure of patent ductus in the presence of pulmonary hypertension carried a higher potential mortality and less prospects of establishing normality. Surgery is much more urgently needed in the presence of pulmonary hypertension because of the probability of progressive changes in the pulmonary vascular system.

The pressure changes on temporary occlusion of the ductus merely indicate hemodynamic significance of the ductus. The presence of a significant left-to-right shunt would be signalled by a favorable response, and the presence of an insignificant shunt by no response. A predominantly right-to-left shunt would cause a rise in the pulmonary artery pressure on temporary occlusion of the ductus.

REFERENCES

1. Bothwell, T. H., VanLingen, B., Whidborne, J., Kaye, J., McGregor, M., and Elliott, G. A.: Patent Ductus Arteriosus With Partial Reversal of the Shunt, Am. Heart J. **44**: 360, 1952.
2. Burchell, H. B., Swan, H. J., and Wood, E. H.: Demonstration of Differential Effects on Pulmonary and Systemic Arterial Pressure by Variation in Oxygen Content of Inspired Air in Patients With Patent Ductus Arteriosus and Pulmonary Hypertension, Circulation **8**: 681, 1953.
3. Cosh, J. A.: Patent Ductus With Pulmonary Hypertension, Brit. Heart J. **15**: 423, 1953.
4. Crafoord, C.: Meeting of the Royal Society of Medicine, Brit. M. J. **1**: 946, 1951.
5. Edwards, J. E.: Pathologic Considerations in Adjustments Between the Systemic and Pulmonary Circulations, Cardiovascular Surgery, Philadelphia, 1955, W. B. Saunders Co., p. 100.
6. Ellis, F. H., Jr., Kirklin, J. W., Callahan, J. A., and Wood, E. H.: Patent Ductus Arteriosus With Pulmonary Hypertension: An Analysis of Cases Treated Surgically, J. THORACIC SURG. **31**: 268, 1956.
7. Glenn, W. W. L., Hampton, L. J., and Goodyear, A. V. N.: Use of Controlled Hypotension in Large Blood Vessel Surgery, A. M. A. Arch. Surg. **68**: 1, 1954.
8. Gross, R. E., and Longino, L. A.: Patent Ductus Arteriosus: Observations from 412 Surgically Treated Cases, Circulation **3**: 125, 1951.
9. Jones, J. C.: The Surgery of Patent Ductus Arteriosus, Ann. Surg. **130**: 174, 1949.
10. Kattus, A. A., and Nuller, W. H., Jr.: Patent Ductus Arteriosus With Reversal of Blood Flow, Ann. Surg. **138**: 870, 1953.
11. Lukas, D. S., Araujo, J., and Steinberg, I.: The Syndrome of Patent Ductus Arteriosus With Reversal of Flow, Am. J. Med. **17**: 298, 1954.
12. Shumaker, H. B., and Lurie, P. R.: Patent Ductus Arteriosus With Pulmonary Hypertension, A. M. A. Arch. Surg. **76**: 179, 1958.
13. Sirak, H. D., and Humphreys, G. H.: The Atypical Ductus: Physiologic and Technical Considerations in Its Surgical Therapy, Surgery **41**: 112, 1957.
14. Smith, G.: Patent Ductus Arteriosus With Pulmonary Hypertension and Reversal Shunt, Brit. Heart J. **16**: 233, 1954.
15. Muller, W. H. J., and Damman, J. F.: The Surgical Significance of Pulmonary Hypertension, Ann. Surg. **136**: 495, 1952.
16. Hultgren, H., Selzer, A., Purdy, A., Holman, E., and Gerbode, F.: The Syndrome of Patent Ductus Arteriosus With Pulmonary Hypertension, Circulation **8**: 15, 1953.
17. Gerbode, F., Holman, E., Hultgren, H., Osborn, J. J., Purdy, A. P., Robinson, S. J., and Selzer, A.: Atypical Patent Ductus, A. M. A. Arch. Surg. **72**: 850, 1956.

Discussion

DR. HARRIS B. SHUMACKER, JR., Indianapolis, Ind.—I think the subject which Dr. Gale has introduced is very timely, since a pessimistic attitude still exists concerning the surgical possibilities for patients who have pulmonary hypertension associated with patent ductus arteriosus. As in the case in his experience, we too do not know how many of our patients have had pulmonary hypertension, for in the earlier days not only were patients with typical signs of patent ductus not subjected to preoperative catheterization, but pressures were also not determined at operation. We have, however, had experience with the treatment of 50 patients who had proved hypertension of some degree.

Of these, 19 had mild pulmonary hypertension, with pulmonary artery pressures of less than 50 mm. Hg systolic. In this group there was one death, an unfortunate cardiac arrest occurring during bronchoscopy carried out postoperatively because of the presence of atelectasis. That patients with mild pulmonary hypertension may have a lesion, which is by no means innocuous, is illustrated by the first slide which shows the very common occurrence of marked cardiomegaly.

In this group of 50 patients, there were 9 in whom the systolic pressure in the pulmonary artery varied between 50 and 60. These patients all did well.

There remains then a group of 22 patients in whom the pressure was systemic in the pulmonary circuit, or nearly so; patients in whom the pulmonary artery pressure was in excess of 80 mm. Hg, or 80 per cent of the systemic in cases in which the systemic pressure was in a hypotensive range. Unfortunately, we are unable to state how many of them had reversal of flow or the extent of reversal of flow in any given case. We do know, however, that many of them had systemic pressure in the pulmonary artery and that, in at least 2, the flow was predominantly from the pulmonary artery to aorta. In 1 case this was evident from the clubbing of the fingers of the left hand and both feet, and in another instance predominant reversal of flow was demonstrated by angiocardiography with injection of dye into the pulmonary artery segment. Both these patients did well. Incidentally, both had a slight drop in pulmonary artery pressure on clamping the ductus, and no decrease in the aortic pressure. In this group of 22 patients with marked pulmonary hypertension there were two deaths. One was due to a technical error, the only one in the entire group. The other occurred in a patient with systemic pressure in the pulmonary artery, who was apparently making an uneventful recovery after division of the ductus when she died suddenly on the fourth day of a cause which could not be explained on clinical grounds or from post-mortem study.

In this group of 50 patients there were 2 who had an unusual difficulty which I would like to illustrate with this slide. These patients do not have marked cardiomegaly but instead have only moderate cardiac enlargement. They have a large main pulmonary artery segment with some enlargement of the proximal portions of the right and left pulmonary arteries, but without significant enlargement of the peripheral branches of the pulmonary arteries. Apparently in this group, in contrast to the usual case, the normal fetal pulmonary resistance persists from the time of birth. The only two deaths in this group of 22 with marked pulmonary hypertension were cases of this sort, the only two of this kind we have treated surgically. Perhaps this group may prove a more difficult problem than the other in which we assume that pulmonary vascular sclerosis gradually develops as the result of increased flow through the pulmonary circuit.

We too have felt, particularly since the one tragic operative death to which I referred, that every effort should be made to gain control of the parent vessels in operating on a patent ductus, and we use the illustrated technique in all cases, no matter how simple, isolating the aorta above and below the ductus and, whenever the ductus is large and pulmonary hypertension exists, dividing the ductus in stages, suturing both sides as the division is completed.

I do not know whether in cases of very long-standing patent ductus with reversal of flow, the patient will subsequently undergo improvement in the pulmonary vascular sclerosis, but I do know that this does occur in some children. It would be my feeling

that no patient should be considered categorically inoperable because of evidence of systemic pressure in the pulmonary circuit nor reversal of flow. It would be my feeling that any such patient should be explored. At operation a favorable pressure response may be demonstrated, as occurred in some of our patients and, if not, I would certainly be in favor of providing the patient with a smaller shunt and taking down the larger natural one, a mechanism of handling the problem which Dr. Muller and Dr. Kattus suggested some years ago.

DR. WILLIS J. POTTS, Chicago, Ill.—In keeping with the President's address which included some reference to word derivations, I would like to say something about the word "ductus." The plural of ductus is ductus. Those of you who were exposed to Latin will remember the fourth declension—the key word in my book was fructus. Ductus belongs in the same category and we properly speak of one ductus or a hundred ductus. It sounds a bit unfamiliar but that's the way it is to those who take their Latin seriously.

Pulmonary hypertension as a complication of patent ductus is a serious problem. Our experience parallels that of the speaker. Interestingly enough, if at operation the pulmonary pressure drops a little, as much as 10 mm. Hg, when the ductus is clamped, the child will do well after division and suture. On the contrary, if pulmonary pressure goes up as much as 10 mm. Hg when the ductus is clamped, results after division and suture will be bad.

We have operated upon a number of children with increased pulmonary pressure but in only 5 did the pressure go up in the pulmonary artery when the ductus was clamped. The ages of these children varied from 4 to 12 years. Knowing that the future of a child with severe hypertension is rather hopeless we divided the ductus in all 5 patients. Our hopes that reversal of pressure might occur because of the age of our patients were not realized. Two died suddenly, one 6 months, the other 2 years after surgery. Two are cardiac cripples in and out of decompensation and constantly on digitalis. The fifth child, aged 12 years, had equal systemic and pulmonary pressure and died on the operating table of hemorrhage. The pulmonary artery was 2.5 inches—not centimeters—in diameter, the ductus approximately 15 mm. in diameter. I put a clamp on the ductus and shortly thereafter the pulmonary artery ruptured. That was a mistake. What I should have done was exactly what was suggested today—an aortic clamp (Potts-Smith) should have been placed around the aorta to occlude the aortic end of the ductus close to the aortic wall. A ductus clamp then could have been placed on the pulmonary end of the ductus without causing tension and rupture. We pay so dearly for our mistakes.

Incidentally, this has been our only operative or hospital death in 495 cases of surgical occlusion of patent ductus. When a discussion of mortality comes up I note in the surgical staff an ill-concealed look of satisfaction that this lone catastrophe should have happened to me.

DR. YOUNG (Closing).—I want to thank Dr. Shumacker and Dr. Potts for their remarks. We have felt that the patients who died after their ductus were closed, and they had no change in pressures on occlusion of their ductus, died because the lesions corrected were not hemodynamically significant. Two of these patients had septal defects in addition to the ductus. The significant lesions probably were left uncorrected.

Our catheterization laboratory has found only 1 patient with patent ductus arteriosus and a marked reversal of flow. She was not subjected to surgery.

This slide shows an indicator dilution curve with evidence of a right-to-left shunt. The interrupted slope of the initial deflection in the cuvette recording from the femoral artery is due to the right-to-left shunt.

With control of polycythemia, and then repeating cardiac catheterization, a few more patients might be benefited by closure of the ductus, when they had predominantly right-to-left shunts prior to control of the polycythemia.

ANEURYSMS OF THE THORACIC AORTA

Analysis of 179 Patients Treated by Resection

Michael E. DeBakey, M.D., Denton A. Cooley, M.D., E. Stanley Crawford, M.D. (by invitation), and George C. Morris, Jr., M.D. (by invitation), Houston, Texas

DURING the 7-year period since our first successful resection of an aneurysm of the thoracic aorta, this method of therapy has been employed in 179 cases. This report is concerned with certain observations derived from an analysis of this experience, particularly in relation to methods of resection and to factors bearing upon risk of operation. Although the underlying principle of therapy consisting essentially in excision of the aneurysm is similar for all cases, the method of its application varies in accordance with the type and location of the lesion. Accordingly, the cases are classified into four categories, namely, aneurysms of the descending thoracic aorta, aneurysms of the aortic arch, thoracoabdominal aneurysms, and dissecting aneurysms.

ANEURYSMS OF THE DESCENDING THORACIC AORTA

This group, comprising 73 cases, represents the largest in the series and reflects the relative frequency of involvement of this segment of thoracic aorta. The great majority of these aneurysms were fusiform in character, varying in size from relatively small, well-localized lesions to large, extensive aneurysms that involved virtually the entire descending thoracic aorta (Figs. 1 and 2). Their site of involvement was also variable, although approximately 90 per cent were located in the proximal portion of the descending thoracic aorta. Contrary to earlier reports that syphilis is the most common etiological factor for aneurysms of the thoracic aorta, in this series arteriosclerosis was the most common cause and syphilis was second in frequency, the respective incidences being 47 per cent and 32 per cent (Table I).

The age of these patients ranged from 10 months to 70 years, but most of them, 83 per cent, were in the fifth to seventh decades. The male sex was predominant in a ratio of about 3 to 1.

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In the application of excisional therapy for aneurysms located in this portion of the aorta, the most important consideration stems from the necessity for temporary arrest of aortic circulation through the segment to be resected and the consequent effect of increased vascular resistance upon the

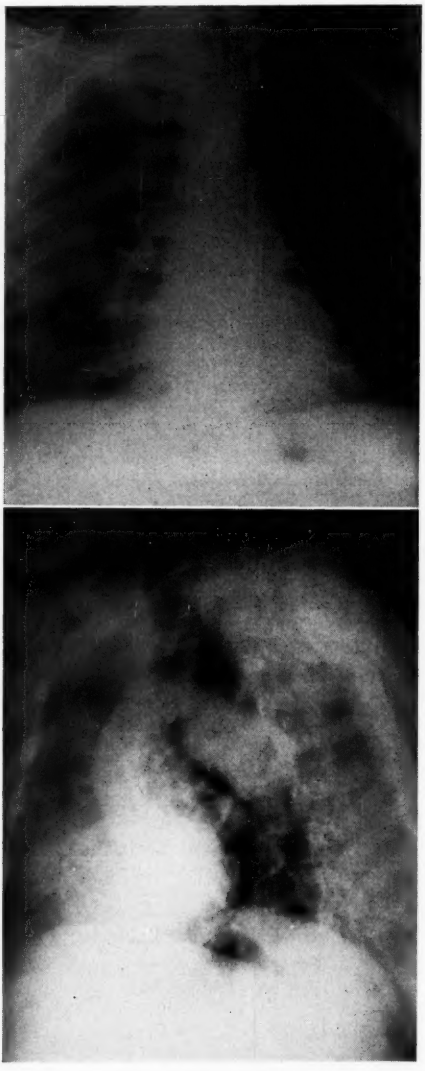


Fig. 1.—*A*, Roentgenogram of chest showing rounded shadow, outlined by peripheral rim of calcification, projecting into left lung field from mid-mediastinal region representing traumatic aneurysm of proximal segment of descending thoracic aorta. The esophagus which is demonstrated by swallowed barium is displaced to right by aneurysm.

B, Angiocardiogram of same patient showing precise location and extent of aneurysm.

heart and ischemic damage upon the spinal cord. Accordingly, control of these factors, and particularly prevention of spinal cord damage, assumes considerable significance. This may be achieved by several methods, including hypothermia, which in our early experience was the primary means used for

C.

D.

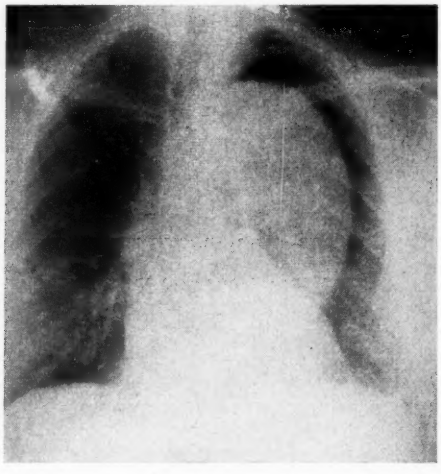
Fig. 1.—Cont'd. *C*, Photograph of same patient made at operation showing characteristic gross appearance of traumatic fusiform aneurysm of proximal portion of descending thoracic aorta following its mobilization from surrounding structures with umbilical tapes encircling aorta above (at right) and below aneurysm.

D, Photograph in same patient made at operation showing completed replacement of resected segment of aorta and aneurysm with knitted, crimped Dacron "graft."

TABLE I. FUSIFORM ANEURYSMS OF DESCENDING THORACIC AORTA
MORTALITY ACCORDING TO ETIOLOGY

ETIOLOGY	NO. CASES	DEATHS	
		NO. CASES	PER CENT
Arteriosclerosis	34	12	35
Syphilis	23	4	17
Trauma	9	0	0
Undetermined	7	1	14
Total	73	17	23

this purpose. Although in general, hypothermia was found to be effective in preventing neurological sequelae, it was associated with certain well-recognized disadvantages indicating the need for a better method. This led to the development of the method of controlled extracorporeal circulation consisting essentially in the use of a pump-bypass in which oxygenated blood is

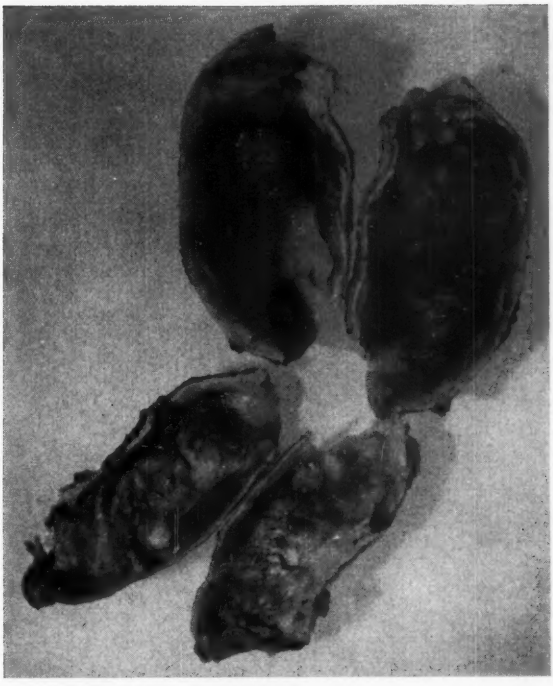


A.

B.

Fig. 2.—*A*, Roentgenogram of chest of 59-year-old white male patient showing large oval shadow projecting from mediastinum into left lung field, representing extensive arteriosclerotic fusiform aneurysm involving virtually entire descending thoracic aorta.

B, Angiocardiogram in same patient showing aneurysm arising just distal to left subclavian artery and extending down to involve distal portion of descending thoracic aorta with pronounced elongation and kinking of aorta.



C.

D.

Fig. 2.—Cont'd. *C*, Photograph of sagittal section of specimen removed at operation in same case showing gross pathologic features of arteriosclerotic fusiform aneurysm. Specimen is shown in two parts because its removal at operation was facilitated by dividing it in this manner.

D, Photograph made at operation in same patient showing completed replacement of resected segment of aorta and aneurysm with Dacron graft (proximal anastomosis is to right).

removed from the left auricle and pumped into the aorta distal to the occluding clamps through a plastic cannula inserted upward into the left femoral artery[4] (Fig. 3). Immediately before insertion of these cannulas, heparin (1.0 mg./Kg. body weight) is administered intravenously and, after completing the procedure of resection and graft replacement, a counteracting dose of protamine sulfate is administered. During the period of occlusion the rate of flow is adjusted from time to time to maintain blood pressure in the arm approximately 10 to 20 mm. Hg above normal systolic pressure. In

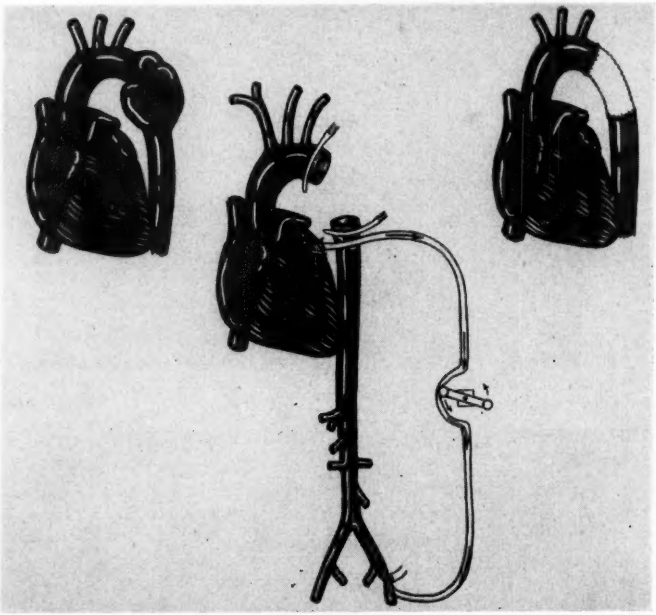


Fig. 3.—Drawing illustrating operative procedure of resection of fusiform aneurysm of descending thoracic aorta using method of controlled extracorporeal circulation to combat ischemic effects of temporary arrest of aortic circulation during performance of procedure. Oxygenated blood is removed from left auricle by means of plastic catheter inserted through appendage and pumped into distal aorta through catheter inserted into left common femoral artery.

general it was found that on this basis flow rates ranging from about 1,200 to 2,500 c.c. per minute were provided. While optimum rates of flow have not been well established, our studies on renal function would suggest that it is desirable to provide sufficient volume of flow in the distal aortic segment to maintain pressure at or above 32 mm. Hg, since demonstrable renal function disappears below this level.[14] Such levels of pressure may be obtained in most instances by flow rates above 20 c.c. per kilogram of body weight.

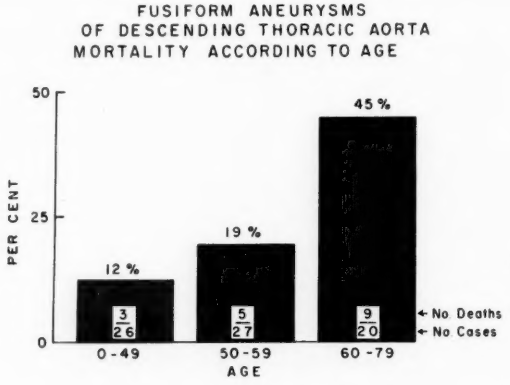
This method was found to be more satisfactory than those previously employed and for this reason it has been applied in the last 36 cases in this series. This conviction is supported by a comparison of the results obtained in this group of cases with those in the earlier series of 37 cases in which other

methods, including hypothermia, were employed. Thus, the mortality was reduced from 27 per cent in the earlier series of 37 cases to 19 per cent in the more recent series of 36 cases. In both series of cases, however, there were 2 patients in whom some manifestations of spinal cord damage occurred. The occurrence of the neurological disturbances in these 4 patients remains a rather puzzling phenomenon. This is particularly true for the 2 patients in the more recent series since the extent of segment resected, the period of occlusion, and the pump bypass flow rates were not significantly different from the other cases in which no neurological disturbances occurred. To add further to the difficulty of explaining this phenomenon is the fact that no such neurological disturbances occurred in 1 patient in this group in whom resection and graft replacement was performed for a fusiform aneurysm of the proximal segment of the descending aorta under normothermic conditions and without pump-bypass. In this case, the aneurysm ruptured during the initial dissection and it became urgently necessary to apply occluding clamps to the aorta proximal and distal to the aneurysm. Despite the fact that the aorta was occluded at this high level for 32 minutes, the patient recovered without any neurological disturbances. It would, thus, appear that considerable variations exist in tolerance of the spinal cord to temporary aortic occlusion and that a number of factors play a role in this phenomenon, including variations in the anatomic pattern and segmental distribution of spinal arteries, development of collateral circulation, as well as measures to combat temporary interruption of blood supply to the cord.

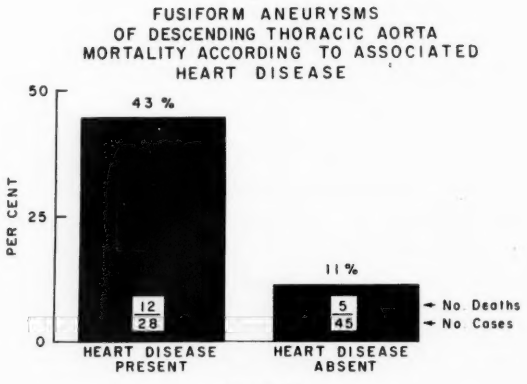
Results of excisional therapy for this type of aortic aneurysm have been increasingly gratifying as exemplified by the progressive decrease in operative mortality rate from 27 per cent in our early experience to 19 per cent in our most recent experience. This observation lends support to the conviction that improvements in the surgical management of these patients, as well as in the actual technical performance of the operation derived from increasing experience, should permit further reduction in the risk. The significance of certain factors in relation to the mortality is illustrated by further analysis of our experience. Among these factors, advancing age, etiology, hypertension, and pre-existing heart disease are particularly significant. The fatality rate, for example, in patients in the seventh and eighth decades of life was more than 2 to 3 times greater than that of younger patients (Graph I). The significance of etiology is demonstrated by the fact that the mortality among patients with aneurysms of arteriosclerotic origin was more than twice that of patients with aneurysms of other causes (Table I). To some extent this may be attributable to the fact that the patients with arteriosclerotic aneurysms were older than the others. The importance of hypertension is demonstrated by the fact that the risk in this group of patients was two times greater than that in the normotensive series (Table II). Similarly the mortality rate in patients with pre-existing heart disease was almost four times greater than that in patients without heart disease (Graph II). The importance of this factor is further emphasized by the fact that, among the 17 operative deaths,

TABLE II. FUSIFORM ANEURYSMS OF DESCENDING THORACIC AORTA
MORTALITY ACCORDING TO BLOOD PRESSURE

BLOOD PRESSURE	NO. CASES	DEATHS	
		NO. CASES	PER CENT
149/89 or less	39	6	15
150/90 or more	34	11	32
Total	73	17	23

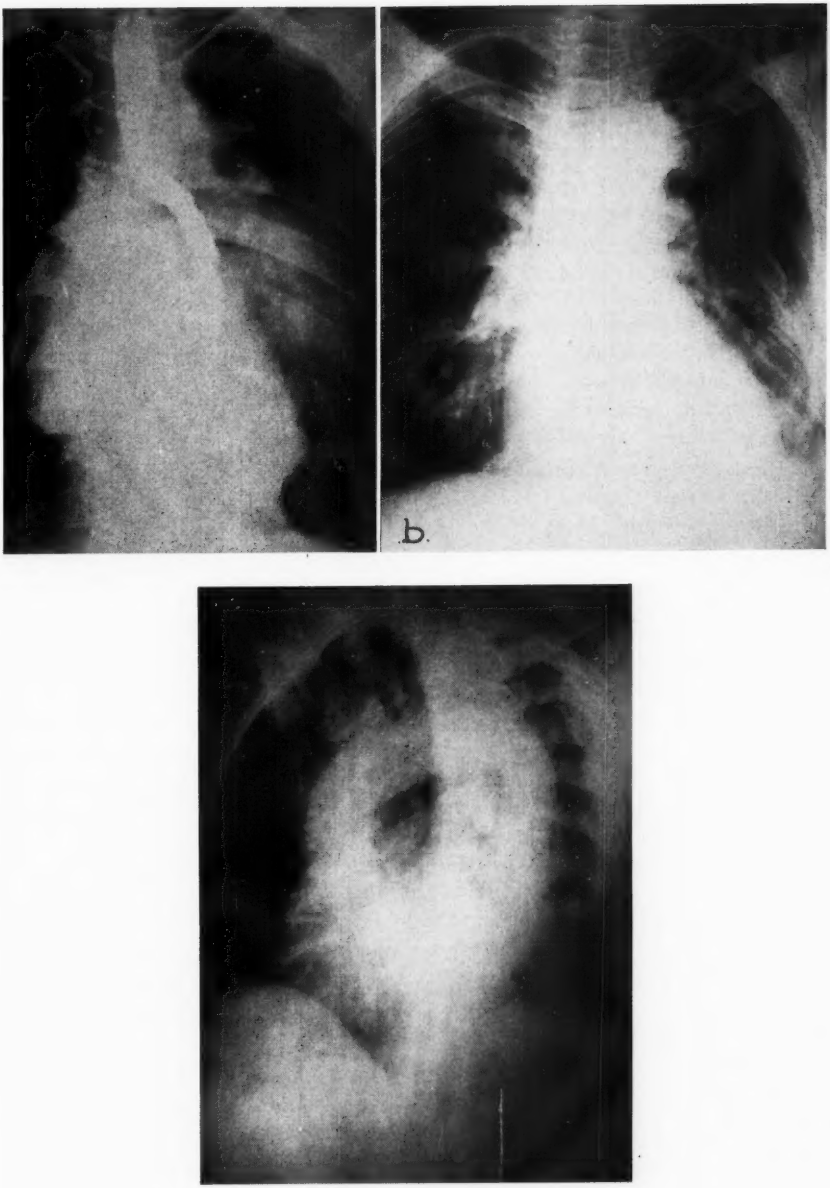


Graph I.



Graph II.

heart failure, mostly in the form of coronary thrombosis, was the most common cause, accounting for 9 fatalities. Among the remaining operative deaths, 2 were attributable to shock, 2 were caused by subsequent rupture of the homograft, 1 to pre-existing septicemia, 1 to massive upper gastrointestinal hemorrhage, and 2 to complications of an associated aneurysm of the abdominal aorta. In one of these latter cases in which the operation was performed under hypothermia, it was noted immediately following completion of the resection of the thoracic aneurysm that all pulses in the lower extremity were



C.

Fig. 4.—*A*, Preoperative roentgenogram of chest in a 50-year-old Negro man showing extensive syphilitic fusiform aneurysm of descending thoracic aorta (case report on this patient was previously presented[2]).

B, Roentgenogram of chest in same patient made approximately 5 years after operation consisting of resection and homograft replacement of aneurysm. Patient has remained well and resumed completely normal activities.

C, Angiocardiogram in same patient made approximately 5 years after operation showing relatively normal outline of descending thoracic aorta.

absent and a saddle embolus of the aortic bifurcation was suspected. Immediate abdominal exploration revealed, however, the presence of a small fusiform aneurysm of the abdominal aorta which had become completely occluded by thrombus, presumably formed during the period of thoracic aortic occlusion. It was resected and replaced by a homograft and, although circulation was restored in the lower extremities, the patient died of renal failure and uremia on the eighth postoperative day. In the other case, the associated aneurysm of the abdominal aorta was diagnosed preoperatively and resection of the aneurysm of the thoracic aorta was performed first. Although the patient's immediate postoperative course was satisfactory, acute rupture of the aneurysm of the abdominal aorta occurred 36 hours later causing rapid death. Of further interest in this connection is the fact that there were 4 other patients in this series who also had an associated aneurysm of the abdominal aorta which was successfully resected at a previous operation.

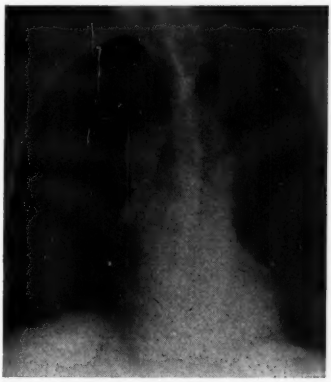
Follow-up observations on all the surviving patients in this group extending for periods ranging from several months to over 5 years provide highly gratifying results. With few exceptions there has been complete relief of symptoms and resumption of normal activities. Of particular interest is the fact that the first 2 patients in this series, in whom operation was performed approximately 5 years ago, have remained well and working.[2, 7, 8] An angiocardiogram performed recently in 1 of these patients whose case report appeared in a previous publication[2] reveals a relatively normal outline of the descending thoracic aorta (Fig. 4). There have been 8 late deaths in this group occurring from a few months to about 1½ years after operation. Three of these deaths were due to heart disease, 1 to cerebrovascular thrombosis, 1 to pneumonia, and 3 to complications of the graft replacement. Among this latter group, 2 were due to aneurysm formation and rupture of the homograft, one 5 months and the other 7 months after operation. In the third case, Ivalon was used as the graft replacement and death occurred 3 months after operation from infection, mediastinal abscess, and secondary hemorrhage. In this connection, it may be significant that none of these graft replacement complications have been encountered since we have employed the crimped, knitted Dacron arterial replacement.

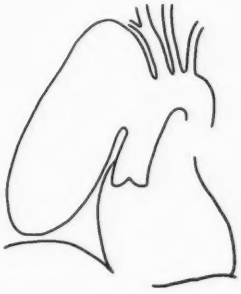
ANEURYSMS OF THE AORTIC ARCH

This group, comprising 50 cases, represents the most serious form of aortic aneurysm not only because of the gravity of this disease but also because of the inherent difficulties involved in the application of excisional therapy. Syphilis was the predominant etiological factor, being present in 35 cases while, in the remainder, the underlying cause was considered to be arteriosclerosis in 10, trauma in 3, and undetermined in 2 (Table III). The ratio of males to females was 4 to 1 and the majority of the patients were in the sixth and seventh decades of life. Although 26 cases were sacciform in type and 24 fusiform, in both instances there was considerable variation in size from relatively small, well-localized lesions to large extensive aneurysms involving virtually the entire arch (Figs. 5, 6, 7, 8).

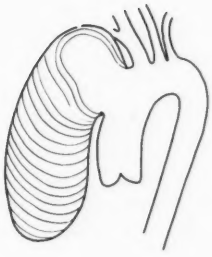
TABLE III. ANEURYSMS OF AORTIC ARCH
MORTALITY ACCORDING TO ETIOLOGY

ETIOLOGY	NO. CASES	DEATHS	
		NO. CASES	PER CENT
Syphilis	35	19	54
Arteriosclerosis	10	8	80
Trauma	3	1	33
Undetermined	2	0	0
Total	50	28	56





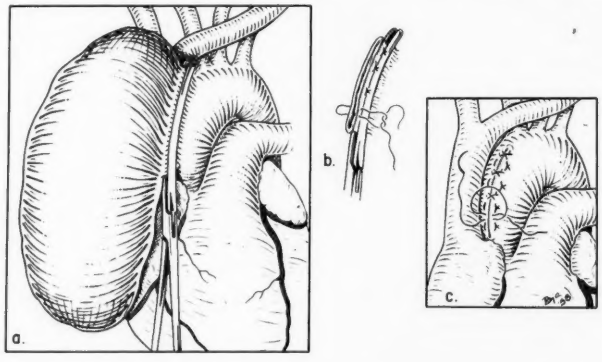
A *I*



B.

Fig. 5.—*A*, Roentgenogram of chest of patient showing large shadow projecting into right lung field, representing extensive sacciform aneurysm with relatively narrow neck arising from ascending aorta as depicted in diagram on right. *B*, Angiocardiogram in same patient showing, by means of radiopaque dye, precise location of neck of sacciform aneurysm arising from ascending aorta. As indicated by diagram on right, most of lumen of sac is filled with thrombus.

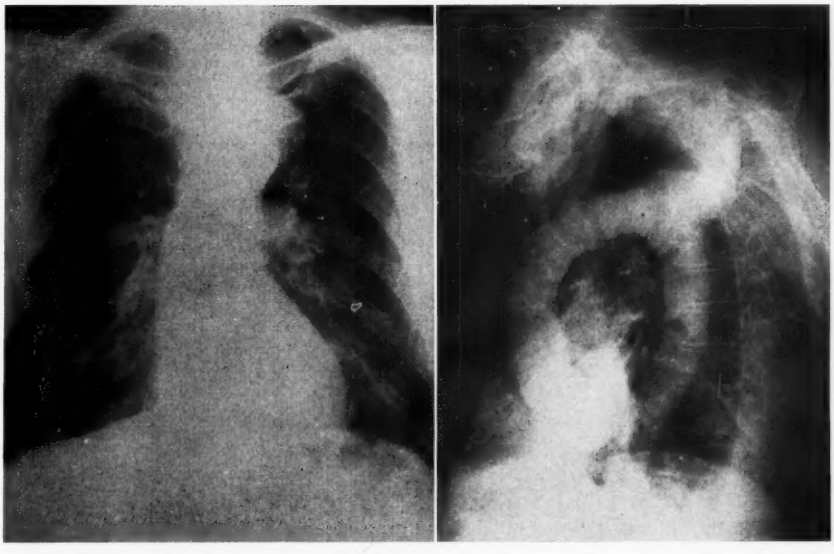
Several methods of excisional therapy may be employed for this group of aneurysms depending upon the location and nature of the lesion. In certain forms of sacciform aneurysms, for example, in which the neck of the sac is relatively small, the procedure of tangential excision and aortorrhaphy may be the most satisfactory[1] (Fig. 5*C*). Even in cases in which the sac itself is quite large and extensive, this procedure may be effectively applied so long as the neck is relatively narrow and involves less than half the circumference of the aorta (Fig. 5). In fusiform aneurysms, however, or in sacciform aneurysms in which the neck of the sac encroaches upon a considerable segment of the aortic circumference, extirpational therapy requires resection of the entire segment of the diseased aorta with graft replacement (Fig. 6). The necessity for temporary arrest of aortic circulation in the performance of this procedure imposes serious problems. For one thing, arrest of circulation at this level interrupts cerebral blood flow which, even after a few minutes, can produce fatal ischemic damage to the central nervous system.



C.

Fig. 5.—Cont'd. *C*, Drawing illustrating procedure of tangential resection and aortorrhaphy which was successfully performed in this patient.

For another, occlusion of the ascending aorta rapidly imposes such a serious strain upon the left ventricle that acute cardiac failure may ensue. To overcome these problems, several methods may be employed depending upon the location of the lesion. Thus, for aneurysms involving the transverse arch or the distal portion of the ascending aorta the use of temporary shunts is considered the procedure of choice (Figs. 7 and 8). In such cases in which the proximal portion of the ascending aorta is uninvolved, it is possible to apply the shunt proximal and distal to the lesion by end-to-side anastomosis, using a partial occlusion clamp on the aorta and thus to perform the procedure without interruption of circulation at any time (Figs. 7 and 8). In our early experience with this bypass principle, the materials used for this purpose were of such nature as to permit their application as temporary shunts only, and following the procedure of resection and graft replacement they were removed.[5, 6] More recently, however, with the development of the crimped Dacron tube, it has become possible to employ this bypass principle



A. *B.*

C. *D.* *E.*

Fig. 6.—*A*, Roentgenogram of chest showing rounded shadow in upper left mediastinum representing sacciform aneurysm of proximal segment of descending thoracic aorta.

B, Angiocardiogram in same patient showing precise location and intraluminal dimensions of sacciform aneurysm.

C, Photograph made at operation in same patient showing gross appearance and anatomic relationship of sacciform aneurysm of descending limb of aortic arch.

D, Photograph of excised segment of aorta involved by the aneurysm.

E, Photograph made at operation in same patient showing completed replacement with Dacron "graft." (This is one of 4 patients with aneurysms of the thoracic aorta in this series who also had successful resection of an aneurysm of the abdominal aorta.)

in a manner that permits its application both as a temporary and a permanent shunt (Figs. 7 and 8). This is a distinct advantage in reducing the extent of the surgical procedure and the hazards of the operation.

In cases in which the aneurysm involves the proximal portion of the ascending aorta excisional therapy requires the use of the cardiopulmonary

A. *B.*

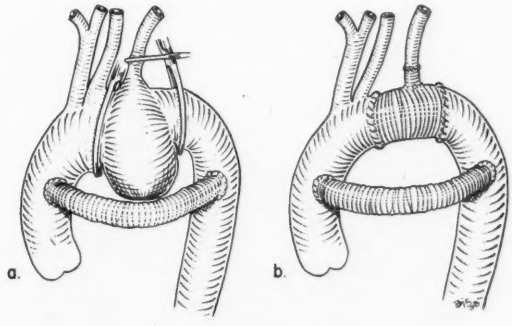
C.

Fig. 7.—*A*, Roentgenogram of chest showing rounded shadow projecting from mediastinum into left lung field representing fusiform aneurysm of transverse arch of aorta.

B, Angiocardiogram showing extent and location of aneurysm.

C, Photograph made at operation showing extent and location of aneurysm involving transverse arch between (*1*) left common carotid artery and (*2*) descending limb of aortic arch.

bypass method by means of an artificial heart-lung apparatus (Fig. 9). Details concerning the application of this method have been described in previous reports.[3, 10]



D.

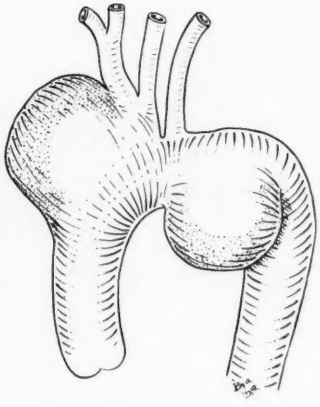
E.

Fig. 7.—Cont'd. *D,* Drawing showing method of temporary bypass from ascending to descending aorta employed for resection of this type of aneurysm.

E, Photograph made at operation showing completed procedure of resection with replacement by Dacron graft including anastomosis to left subclavian artery and use of bypass Dacron graft from ascending aorta to descending limb of aortic arch.

Analysis of results of operation in this group of aneurysms reveals a number of factors having a significant bearing upon the operative risk, including particularly the type and location of the aneurysm, age, and the method of excisional therapy. Aneurysms of arteriosclerotic origin, for example, were associated with a considerably higher operative mortality than other types of lesions, owing probably to the fact that these patients were more advanced in age and were more likely to have associated heart disease (Table IV). The importance of the type and extent of the aneurysm is demonstrated by the fact that the operative mortality in fusiform aneurysms was

A.

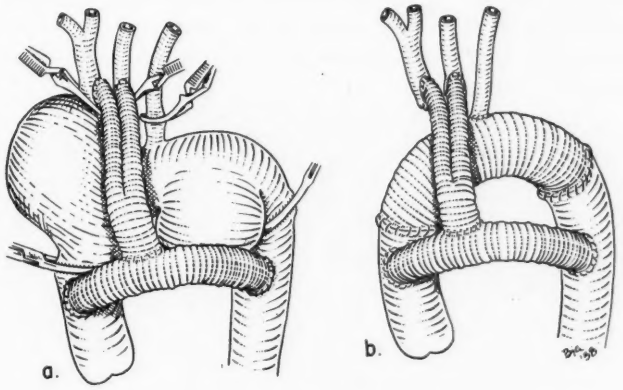


B.

Fig. 8.—*A*, Roentgenogram of chest showing supracardiac mediastinal shadow representing extensive fusiform aneurysms of aortic arch.

B, Angiocardiogram of same patient showing precise location of aneurysms involving distal part of ascending aorta and transverse arch as indicated in drawing on right.

75 per cent, whereas among sacciform aneurysms it was only 28 per cent. The significance of age is indicated by the fact that among the 16 patients in the seventh decade of life the operative mortality was 75 per cent, whereas among the remaining patients below this age it was 47 per cent.



C.

D.

Fig. 8.—Cont'd. *C*, Drawings illustrating method of resection and graft replacement using method of temporary bypass subsequently converted into permanent graft.
D, Photograph made at operation showing completed procedure.

TABLE IV. DISSECTING ANEURYSMS OF THORACIC AORTA
MORTALITY ACCORDING TO AGE

AGE	NO. CASES	NO. DEATHS
30-39	2	0
40-49	13	5
50-59	7	2
60-69	10	2
70-79	2	1
Total	34	10 (29%)

Although the total mortality of 56 per cent in this group of patients is relatively high, it must be evaluated against the background of the extreme gravity of the natural course of this disease. Moreover, there is reason to believe that with increasing experience in the surgical management and with technical improvements in the performance of the operation, the operative risk should be steadily reduced. Among the surviving patients, the results have been highly gratifying with complete relief of symptoms and restoration of normal activity.

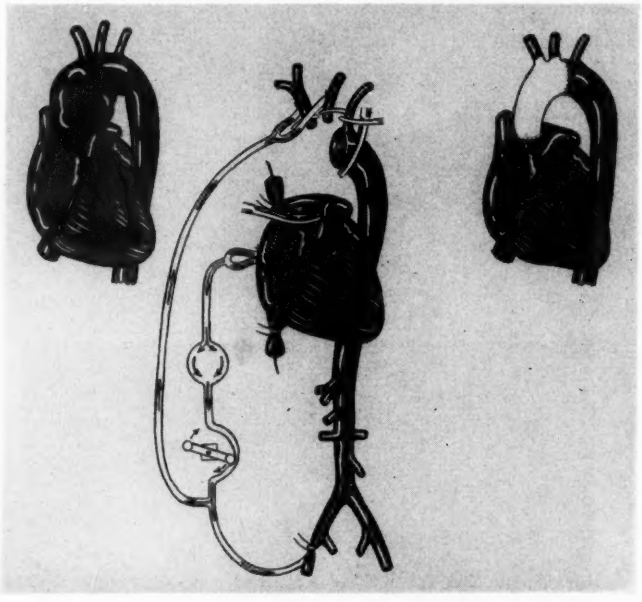


Fig. 9.—Drawing showing method of resection and graft replacement for fusiform aneurysms involving proximal part of ascending aorta using cardiopulmonary bypass by means of artificial heart-lung apparatus.

THORACOABDOMINAL ANEURYSMS

Aneurysms located in this segment of the aorta derive significance from the fact that they involve the principal vessels to the abdominal viscera (Fig. 10). Accordingly, excisional therapy of these lesions is associated with considerable danger of producing fatal ischemic damage to such vital structures as the liver, kidneys, and gastrointestinal tract, as a consequence of temporary arrest of blood flow to these organs during the period of resection and graft replacement.

Syphilis was the predominant cause of this group of aneurysms, being present in 15 of the 22 cases. Among the remainder there were 3 of arteriosclerotic, 2 of traumatic, and 2 of congenital origins. All but 2 of the cases were males, and most of the patients were in the fifth, sixth, and seventh decades of life.

As previously indicated, the most important consideration in extirpation of aneurysms of this type arises from the potential ischemic damage to the abdominal viscera as a consequence of temporary arrest of circulation during performance of the procedure.[11] Moreover, there is reason to believe that the kidneys represent the most sensitive abdominal organs to ischemia. To overcome this problem, several methods may be employed, including hypothermia, controlled extracorporeal circulation, and temporary shunts. The

Fig. 10.—Aortogram showing fusiform aneurysm arising in distal segment of descending thoracic aorta and extending down to involve upper segment of abdominal aorta including celiac, superior mesenteric, and renal arteries.

latter 2 methods were found to be satisfactory in achieving this objective and have been described in previous reports.[4, 11] More recently, still another method has been developed which is considered to have additional advantages (Fig. 11). This method was made possible through the use of our crimped, knitted Dacron tube which permits conversion of the temporary shunt into the permanent graft.

In the application of this method, the Dacron tube, which is provided with branches for the celiac, superior mesenteric, and renal arteries, is attached by end-to-side anastomosis using partial tangential occlusion clamps to the descending thoracic aorta immediately above the aneurysm and by end-to-end anastomosis to the abdominal aorta below the aneurysm (Fig. 11, *a*). The left renal artery is then detached from its aortic origin and the distal end is sutured by end-to-end anastomosis to the corresponding branch on the Dacron tube (Fig. 11, *b*). Following completion of these procedures

the occlusion clamps are released, permitting blood flow through the Dacron tube to the left renal artery and the abdominal aorta distally. By this means circulation to the left kidney is arrested for a period of about 10 or 15 minutes. Circulation through the aneurysm is then arrested by applying occluding clamps to the celiac, superior mesenteric, and right renal artery, as well

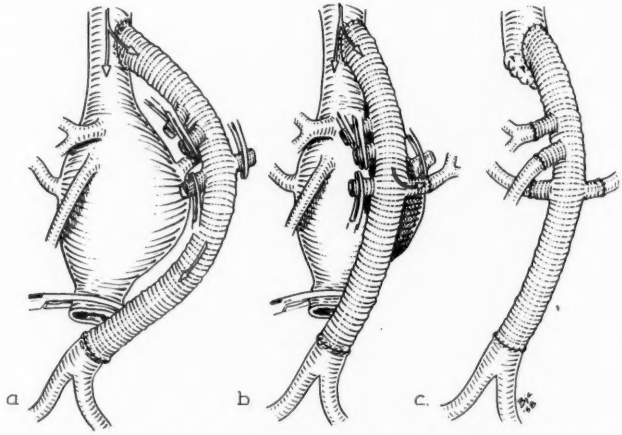


Fig. 11.—Drawing showing method of resection and graft replacement in thoracoabdominal aneurysms using crimped, knitted Dacron tube which permits conversion of the temporary shunt into permanent graft.

a, Dacron tube is attached to descending thoracic aorta above aneurysm by end-to-side anastomosis and to abdominal aorta below aneurysm by end-to-end anastomosis.

b, Left renal artery is attached to corresponding branch on Dacron tube by end-to-end anastomosis and blood flow restored to left kidney. Occluding clamps are then applied to remaining visceral branches and to aorta above aneurysm but below proximal end-to-side anastomosis of Dacron tube, and aneurysm is excised.

c, End-to-end anastomosis is then performed between respective branches of Dacron tube and right renal, superior mesenteric, and celiac arteries, in that order, with restoration of circulation following completion of each anastomosis. Procedure is completed by suture closure of distal end of thoracic aorta immediately below end-to-side anastomosis of Dacron tube.

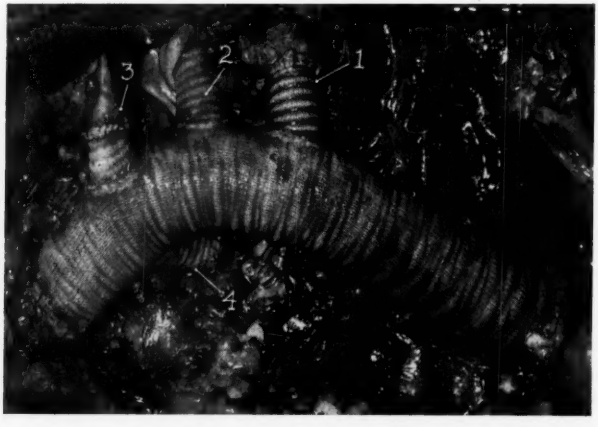
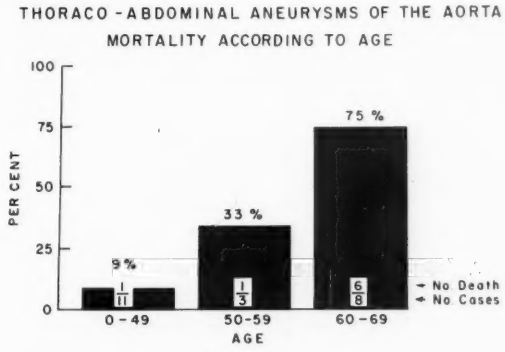


Fig. 12.—Photograph made at operation showing completed replacement of excised segment of thoracoabdominal aorta and aneurysm with Dacron tube, including branches to (*1*) celiac, (*2*) superior mesenteric, (*3*) left renal, and (*4*) right renal arteries.

as to the aorta above and below the aneurysm. After excising the aneurysm, end-to-end anastomoses of the right renal, superior mesenteric, and celiac arteries are performed in that order to the respective branches on the Dacron tube. After completion of each of these anastomoses, circulation is restored by releasing the occluding clamp on the corresponding branch of the Dacron tube. In this manner the period of ischemia to the right kidney should be less than 25 or 30 minutes and to the other arteries about 45 to 50 minutes which, according to our experience, is well within the safe period of tolerance of these organs to anoxia. The Dacron tube which has been serving as a temporary shunt is now converted into a permanent graft by suture closure of the thoracic aortic end just below the end-to-side anastomosis of the Dacron tube (Figs. 11 and 12).



Graph III.

In this series of 22 cases there were 8 operative deaths, 4 of which were caused by heart failure, 1 by renal failure, and 3 by the development of a bleeding syndrome. The significance of age upon operative mortality is well demonstrated by the fact that three fourths of the deaths occurred in patients over 60 years of age (Graph III). The results among the patients who survived operation have been highly gratifying. Follow-up observations extending over periods ranging up to 2½ years reveal maintenance of normal activities.

DISSECTING ANEURYSMS OF THE AORTA

This type of aneurysm represents one of the most serious forms of aortic disease and one of the most challenging problems in the application of surgical treatment, for several reasons. For one thing, the lesion is usually not well localized and often extends throughout the aorta. For another, the general condition of these patients is usually worse than that of others with aortic aneurysms owing presumably to the more acute development of the disease and the frequent association of hypertension and other manifestations of cardiovascular, renal, and respiratory disturbances. This is clearly reflected by studies of the natural course of the disease which have demonstrated its rapidly fatal termination in over 75 per cent of cases.[15]

The methods of surgical treatment for this disease have been reported in a previous publication[9] and have now been employed in 34 cases. The majority of these patients were in the fifth to seventh decades of life and all but 8 were males (Table IV). Moderate to severe hypertension was present in about 70 per cent of cases. Contrary to previous reports,[12, 13, 15] most of which have been based upon necropsy studies, indicating that the diseases usually involve the aortic arch, only 4 cases in our series were of this type. The dissecting process in all the others arose in the proximal segment of the descending thoracic aorta. This discrepancy in the location of the lesion in our surgical cases and in the studies of others based upon necropsy material may be explained by the fact that dissecting aneurysms arising in the ascending aorta are more likely to have a rapidly fatal course. On the other hand, lesions arising in the descending thoracic aorta tend to have a subacute course and thus to permit more time for diagnosis and treatment. In any event, this is a fortunate pathologic feature of the latter group of cases since, as indicated below, it permits application of a more effective form of surgical treatment.

In general, two types of surgical procedure were employed in this series of cases depending upon the nature of the lesion. Thus, in cases in which the dissecting process begins in the ascending aorta, the procedure consists in the creation of a re-entry passage into the true aortic lumen above with obliteration of the false passage below. This is accomplished by complete transection of the descending thoracic aorta between occluding clamps, excising a small segment from the inner wall above to produce re-entry of the outer lumen, approximation of the outer and inner layers below with a continuous suture, and then performance of an end-to-end anastomosis. As indicated previously, this procedure is obviously not curative; its essential objective is to produce re-entry of the false passage into the normal lumen as close to the point of origin of the dissection as may be feasible in order to diminish the likelihood of rupture above and to restore normal aortic circulation below. Among the 4 cases in this series in which this type of procedure was used, there were 2 deaths, 1 from rupture of the dissecting aneurysm into the pericardium and the other from heart failure.

For lesions arising in the descending thoracic aorta, usually just below the level of the left subclavian artery, a more curative type of procedure may be employed (Fig. 13). This is accomplished by excision of the segment involved in the origin of the dissecting process with graft replacement. In some cases in which the lesion is well localized to the descending thoracic aorta, complete resection of the dissecting aneurysm is possible. In other cases in which the dissecting process extends down into the abdominal aorta, the proximal portion involved in the origin of the dissection is excised, the false passage below is obliterated by suture of the inner and outer wall, and aortic continuity restored by graft replacement (Fig. 14). In the performance of this procedure, like that for fusiform aneurysms of the descending thoracic aorta, some method must be employed to combat the ischemic effects

of temporary arrest of aortic circulation. Although hypothermia was originally used for this purpose, more recently we have preferred the method of controlled extracorporeal circulation, as described above, for resection of fusiform aneurysms of the descending thoracic aorta for similar reasons.

This latter method of surgical treatment for dissecting aneurysms was employed in 30 cases in this series with 8 deaths, 4 of which were caused by heart failure and coronary thrombosis, 2 by development of proximal dissection and rupture into the pericardium, 1 by pulmonary embolism, and 1

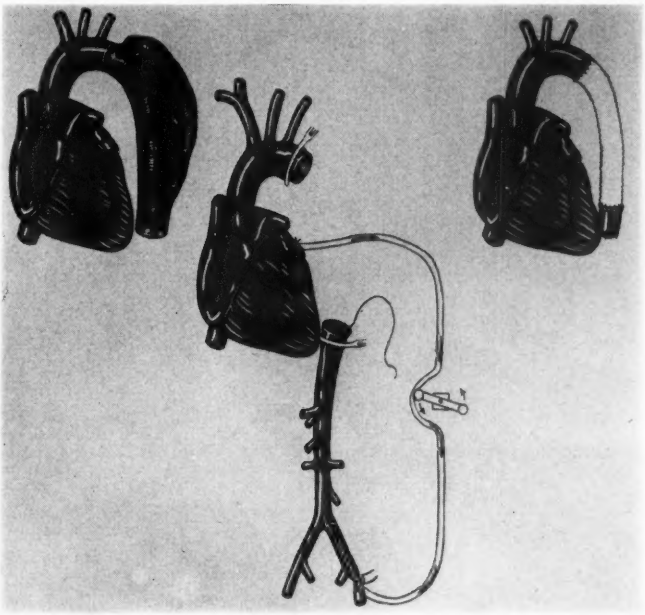


Fig. 13.—Drawing showing method of surgical treatment of dissecting aneurysms of aorta using controlled extracorporeal circulation to provide blood flow into aorta distal to site of application of occluding clamps. After excising segment of dissecting aneurysmal process above its origin, the false lumen in the distal end is obliterated by sewing outer and inner walls together with continuous suture. Anastomosis of graft to upper aortic opening is done by continuous suture with 4-0 arterial silk. Anastomosis of graft to lower opening in aorta with restoration of aortic continuity is then performed.

by ventricular fibrillation during hypothermia. Contrary to other forms of aortic aneurysms, age did not seem to play a significant role in the mortality in dissecting aneurysms (Table IV). Hypertension, however, was found to be highly significant in this regard since there were no deaths in patients considered to be normotensive (Table V).

TABLE V. DISSECTING ANEURYSMS OF THORACIC AORTA
MORTALITY ACCORDING TO BLOOD PRESSURE

BLOOD PRESSURE	NO. CASES	DEATHS	
		NO. CASES	PER CENT
149/89 or less	10	0	0
150/90 or more	24	10	41
Total	34	10	29

With few exceptions, the results obtained in the patients surviving operation have been highly gratifying. Follow-up observations extending up to 3½ years after operation reveal that most of the patients have been able to resume normal activities. There were 2 patients with residual partial paraplegia and 1 late death in this group (occurring about 7 months after operation) from severe uncontrolled hypertension. Subsequent aortographic studies in some cases revealed a relatively normal outline of the descending thoracic aorta (Fig. 14).

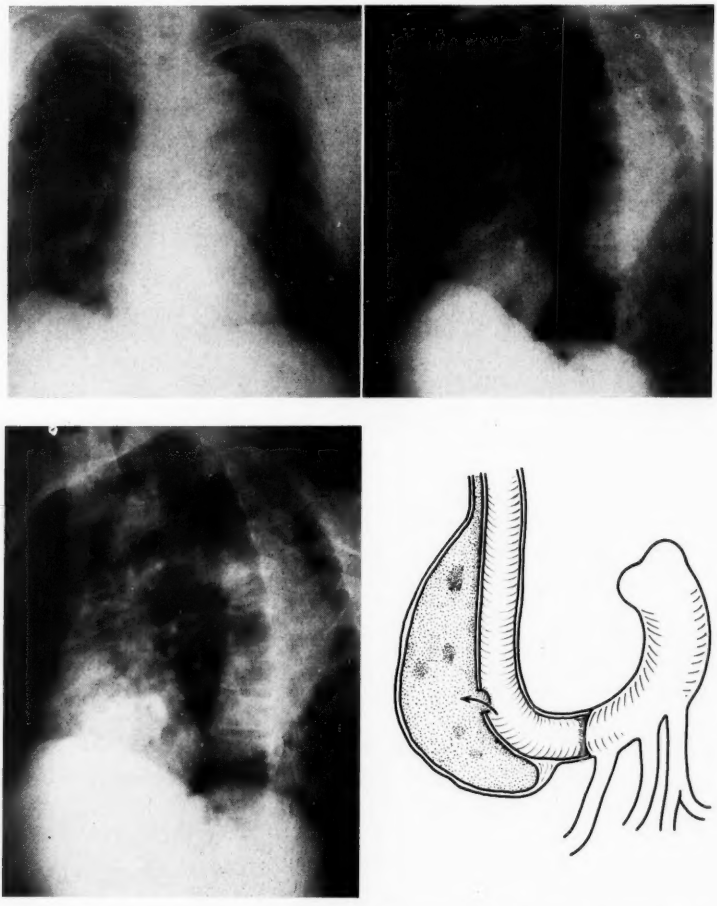
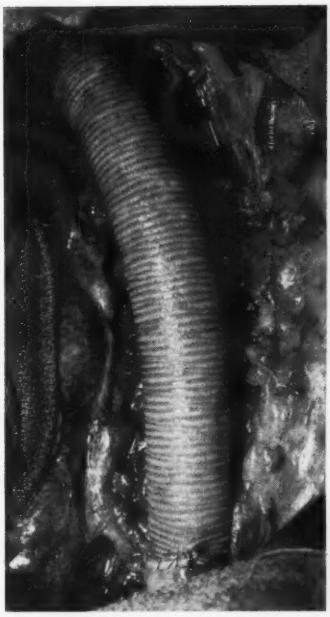


Fig. 14.—*A*, Roentgenograms of chest of patient with dissecting aneurysm of aorta showing, in anteroposterior projection, characteristic widening of supracardiac mediastinal shadow with relative radiolucency in its more peripheral part and, in lateral projection, extensive involvement of descending thoracic aorta.

B, Angiocardiogram of same patient showing characteristic double aortic lumen produced by relative difference in concentration of dye in true and false aortic lumens as depicted in diagram on right.

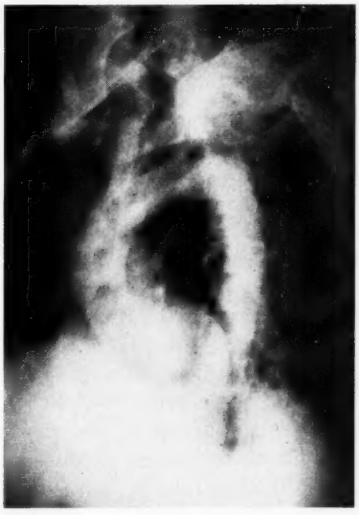
C.

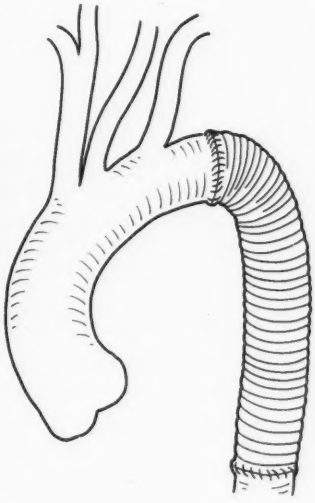


D.

Fig. 14.—Cont'd. *C,* Photograph of sagittal section of resected segment of dissecting aneurysm in same patient showing gross pathologic features of lesion with arrow pointing to intimal tear through which true lumen communicated with false lumen. Lower end of photograph shows cross-sectional view of specimen demonstrating characteristic double-barreled aorta with smaller true lumen and larger false lumen produced by dissection.

D, Photograph made at operation in same patient showing completed replacement of segment of aorta and dissecting aneurysm with Dacron graft.





E.

Fig. 14.—Cont'd. *E,* Angiocardiogram in same patient made approximately 7 months after operation showing relatively normal outline of thoracic aorta. Patient has been completely relieved and has resumed normal activities.

SUMMARY

1. Observations concerning methods and risk of operation are presented from an analysis of 179 cases of aneurysm of the thoracic aorta treated by resection. The cases are classified into 4 groups: (1) aneurysms of the descending thoracic aorta, (2) aneurysms of the aortic arch, (3) thoracoabdominal aneurysms, and (4) dissecting aneurysms.

2. Group 1, comprising 73 cases, represents the largest series and reflects the relative frequency of involvement of this segment. In the application of excisional therapy, the most important consideration stems from the necessity for temporary arrest of aortic circulation through the segment to be resected and the consequent effect of increased vascular resistance upon the heart and ischemic damage upon the spinal cord. Hypothermia was for some time the primary means used for prevention of neurological sequelae but was associated with certain disadvantages. Consequently, a method of controlled extracorporeal circulation was developed consisting of a pump-bypass in which oxygenated blood is removed from the left auricle and pumped into the aorta distal to the occluding clamps through a plastic cannula inserted upward into the left femoral artery. This method has been applied in the last 36 cases in this series and the mortality has been reduced from 27 per cent in the earlier series of 37 cases to 19 per cent in this more recent series. Owing to the fact that, in each series, 2 patients suffered some degree of neurological disturbance unexplainable by any variable in method or condition of the patient, the authors conclude that considerable variations exist in tolerance of the spinal cord to temporary aortic occlusion. Certain factors, such as age, etiology, hypertension, and pre-existing heart disease, are significant in the mortality rate. Follow-up observations extending for periods ranging from several months to over 5 years provide highly gratifying results, revealing with few exceptions complete relief of symptoms and resumption of normal activities.

3. Group 2, comprising 50 cases, represents the most serious form of aortic aneurysm because of the gravity of the disease and the inherent difficulties involved in excisional therapy. In certain sacciform aneurysms of the aortic arch, the procedure of tangential excision and aortorrhaphy may be preferable, but in fusiform aneurysms and in sacciform aneurysms in which the neck of the sac is relatively large, resection of the entire segment with graft replacement is necessary. Temporary arrest of aortic circulation at this level interrupts cerebral blood flow and can produce fatal ischemic damage to the central nervous system. Furthermore, a serious strain is thus placed upon the left ventricle causing the possibility of acute cardiac failure. Several methods may be employed to overcome these problems. For aneurysms involving the transverse arch of the distal portion of the ascending aorta, use of a temporary shunt is considered the procedure of choice. Recently, with the development of the crimped Dacron tube it has been possible to apply the bypass principle in a manner that permits its application both as a temporary and a permanent shunt. In cases in which the proximal portion of the ascending aorta is involved, cardiopulmonary bypass by means

of an artificial heart-lung apparatus is required. Factors bearing upon the operative risk include type and location of the aneurysm, age, and method of excisional therapy. The total mortality in this group was 56 per cent, but this figure must be evaluated against the extreme gravity of the natural course of this disease. Among surviving patients the results have been highly gratifying.

4. Aneurysms in Group 3 are significant particularly because they involve the principal vessels to the abdominal viscera, and excisional therapy is associated with danger of producing fatal ischemic damage to the liver, kidneys, and gastrointestinal tract as a consequence of temporary arrest of blood flow to these organs. In this group of 22 cases, syphilis was the predominant cause of the lesion. Several methods, including hypothermia, controlled extracorporeal circulation, and temporary shunts, have been employed to overcome the danger of damaging the sensitive abdominal organs. Recently another method has been developed through the use of the crimped, knitted Dacron tube which permits conversion of the temporary shunt into the permanent graft. In this series there were 8 operative deaths, 6 of which occurred in patients over 60 years of age. Results among patients who survived have been excellent, and follow-up observations extending over periods ranging up to 2½ years reveal maintenance of normal activity.

5. Among the 34 cases of dissecting aneurysm, 4 involved the aortic arch and the remainder arose in the proximal segment of the descending thoracic aorta. In cases in which the dissecting process begins in the ascending aorta, the procedure consists in the creation of a re-entry passage into the true aortic lumen above with obliteration of the false passage below. This procedure is obviously not curative; its objective is to diminish the likelihood of rupture above and to restore normal aortic circulation below. For lesions arising in the descending thoracic aorta a more curative type of procedure may be employed, consisting of excision of the segment involved in the origin of the dissecting process and replacement with graft. In cases in which the dissecting process is not well localized, the proximal portion is excised and the false passage below is obliterated by suture of the inner and outer wall. Aortic continuity is then restored by graft replacement. Thirty cases have been treated in this manner with 8 deaths. Age did not seem to be a significant factor in the mortality in dissecting aneurysms, but hypertension was highly significant, since there were no deaths in patients considered to be normotensive. Follow-up observations extending up to 3½ years reveal that most of the patients surviving operation have been able to resume normal activities.

REFERENCES

1. Cooley, D. A., and DeBakey, M. E.: Surgical Considerations of Intrathoracic Aneurysms of the Aorta and Great Vessels, Tr. Southern Surgical Assoc. **63**: 91, 1951; Ann. Surg. **135**: 660, 1952.
2. Cooley, D. A, and DeBakey, M. E.: Resection of the Thoracic Aorta With Replacement by Homograft for Aneurysms and Constrictive Lesions, J. THORACIC SURG. **29**: 66, 1955.
3. Cooley, D. A., and DeBakey, M. E.: Resection of Entire Ascending Aorta in Fusiform Aneurysm Using Temporary Cardiac Bypass, J. A. M. A. **162**: 1158, 1956.

4. Cooley, D. A., DeBakey, M. E., and Morris, G. C., Jr.: Controlled Extracorporeal Circulation in Surgical Treatment of Aortic Aneurysm, Ann. Surg. **146**: 473, 1957.
5. Cooley, D. A., Mahaffey, D. E., and DeBakey, M. E.: Total Excision of the Aortic Arch for Aneurysm, Surg., Gynec. & Obst. **101**: 667, 1955.
6. Creech, O., Jr., DeBakey, M. E., and Mahaffey, D. E.: Total Resection of the Aortic Arch, Surgery **40**: 817, 1956.
7. DeBakey, M. E., and Cooley, D. A.: Successful Resection of Aneurysm of Thoracic Aorta and Replacement by Graft, J. A. M. A. **152**: 673, 1953.
8. DeBakey, M. E., and Cooley, D. A.: Successful Resection of Aneurysm of Distal Aortic Arch and Replacement by Graft, J. A. M. A. **155**: 1398, 1954.
9. DeBakey, M. E., Cooley, D. A., and Creech, O., Jr.: Surgical Considerations of Dissecting Aneurysm of the Aorta, Ann. Surg. **142**: 586, 1955.
10. DeBakey, M. E., Crawford, E. S., Cooley, D. A., and Morris, G. C., Jr.: Successful Resection of Fusiform Aneurysm of Aortic Arch With Replacement by Homograft, Surg., Gynec. & Obst. **105**: 656, 1957.
11. DeBakey, M. E., Creech, O., Jr., and Morris, G. C., Jr.: Aneurysm of Thoracoabdominal Aorta Involving Celiac, Superior Mesenteric, and Renal Arteries. Report of Four Cases Treated by Resection and Homograft Replacement, Ann. Surg. **144**: 549, 1956.
12. Gore, I., and Siewert, V. J.: Dissecting Aneurysm of the Aorta: Pathologic Aspects. An Analysis of Eighty-Five Fatal Cases, Arch. Path. **53**: 121, 1952.
13. Halpert, B., and Brown, C. A.: Dissecting Aneurysm of the Aorta, A.M.A. Arch. Path. **60**: 378, 1955.
14. Morris, G. C., Jr., Witt, R. R., Cooley, D. A., Moyer, J. H., and DeBakey, M. E.: Alterations in Renal Hemodynamics During Controlled Extracorporeal Circulation in the Surgical Treatment of Aortic Aneurysm, J. THORACIC SURG. **34**: 590, 1957.
15. Shennan, T.: Dissecting Aneurysms, Medical Research Council Report Series, No. 193, London, H. M. S. O., 1934.

DISCUSSION

DR. JOHN W. STRIEDER, Brookline, Mass.—I rise only to emphasize a very small segment of Dr. DeBakey's magnificent presentation, namely, the pump bypass. As I mentioned this morning, Dr. James Dow and Dr. James Dickson at the Boston City Hospital Surgical Research Laboratory have been interested in this aspect of hemodynamics.

We have applied the pump which is similar to Dr. DeBakey's, except that it is operated by compressed air and permits one to have a bypass and/or a pump working electively, alternately.

In addition to its use for the excision of thoracic aneurysms, it may also be used in any situation where occlusion of the thoracic aorta is necessary.

We have used it, therefore, in a patient who was severely ill, the result of aortic regurgitation, where we contemplated inserting a Hufnagel valve.

This slide shows the pressures which were taken above and below the point of occlusion through the axillary artery and the femoral artery. Catheters have been passed through both these vessels a suitable distance. One can then see graphically that when the bypass is open with the pump not working, the pressures above and below the point of occlusion of the aorta show a definite differential with the pressures below the point of occlusion falling off, but remaining at approximately 60 mm. Hg systolic. When the bypass is clamped and the pump started, however, the pressure in the distal aorta remains at a satisfactory level, approximately that of the pressure in the proximal aorta. This permits a leisurely approach to the operation described by Hufnagel.

As I mentioned before, it was used in one such patient and was highly successful technically, although subsequently the patient died for reasons which were not discernible at autopsy and apparently bore no relation to the method.

EXPERIENCES WITH THE DAVILA-GLOVER PURSE-STRING SUTURE IN THE CORRECTION OF MITRAL INSUFFICIENCY: A CRITICAL APPRAISAL

Edward M. Kent, M.D., William B. Ford, M.D., John F. Neville, Jr., M.D. (by invitation), and Don L. Fisher, M.D. (by invitation), Pittsburgh, Pa.

DURING the Thirty-fifth Annual Meeting of the American Association for Thoracic Surgery, Davila and Glover and their associates[1] described a method of accomplishing circumferential suture of the mitral ring for the correction of valvular insufficiency. That presentation included their experiences during the perfection of the technique in the experimental laboratory and the use of the operation in the surgical management of 7 patients whose valvular disease was characterized by predominant mitral insufficiency. Although the mortality rate was high in this preliminary report, the authors felt that the results in the surviving patients were most encouraging.

The essentials of the procedure as described by Davila and co-workers consist of placing the suture medially by approaching the posterior aspect of the heart and introducing the needle at a point just to the left of the posterior descending coronary artery and immediately beneath the coronary sinus and distal circumflex coronary artery. The needle then was passed ventrally into the base of the interatrial septum and thence into the right atrium, entering this chamber at a point just ventrad to the ostium of the coronary sinus, then advanced adjacent to the right side of the septum, piercing the anterior right atrial wall at its junction with the anterior base of the septum. At this point, the needle and suture emerged into the transverse sinus and the segment of the suture which was to lie in the transverse sinus was encased in a pericardial sleeve, 4 cm. in length. A tunnel was then made anteriorly beneath the circumflex coronary and great cardiac vein at a point about 1 cm. lateral to the anterior descending coronary. The segment of suture which had been covered by pericardium was then drawn back into the transverse sinus by traction on the posterior "tail." The anterior "tail" was then passed beneath the circumflex coronary through the previously prepared tunnel. The posterior "tail" of the suture was then threaded on a large curved needle and stitched anterolaterally

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Read at the Thirty-eighth Annual Meeting of The American Association for Thoracic Surgery at Boston, Mass., May 16-18, 1958.

into the atrioventricular groove, underpassing the lateral branches of the circumflex coronary so that it finally lay just beneath the circumflex bundle. The two tails were then approximated about 1 cm. to the left of the anterior descending coronary and immediately below the circumflex coronary where they were tied in a double square knot. As this knot is tied, the operator maintains a tactile vigil over the effect on the valvular regurgitation by means of the exploring finger which remains within the left atrium by way of its appendage. This finger also serves as a means to prevent the creation of stenosis of the valvular orifice during the tying of the knot in the purse-string suture and has the equally great responsibility of monitoring the placement of the purse-string suture to ensure against the insertion of any portion of the suture within the left atrial chamber.

It is apparent to all interested observers that there is a great need for a method which will safely and effectively control mitral insufficiency when it is of major proportions. Our purpose in this communication is to present our experiences and results with the use of the Davila-Glover circumferential suture of the mitral ring as a means of attempting to control predominant mitral regurgitation in 30 patients.

METHODS

Patients were selected for surgical correction of mitral insufficiency on the basis of clinical evaluation and supporting data obtained by means of simultaneous right and left heart catheterization and by means of the injection of Evans blue dye into the left ventricle followed by immediate recovery of the medium from the left atrium.

In the area of clinical evaluation of patients, the measures adopted did not differ from those employed generally. The presence of an apical systolic murmur has been the cardinal method of spotting patients suspected of having mitral insufficiency. Some of these patients displayed qualities inherent in their murmurs which are commonly accepted as indicative of a significant degree of valvular incompetence (i.e., loudness, transmission to the axillary area, decreased intensity of the mitral first sound).

Further documentation includes evidence of left ventricular enlargement by means of electrocardiography, roentgenography and fluoroscopy. Additionally, evidence of left atrial enlargement was sought by the same avenues of study. The presence or absence of atrial fibrillation was demonstrated in all instances.

Each patient in this study was severely disabled by predominant mitral insufficiency.

The supporting data which have been obtained by means of combined right and left heart catheterization are available for all save 2 patients. The criteria obtained by these measures include the appearance of a tall regurgitant pulse in the left atrium (V wave exceeding the height of the C wave by 10 mm. Hg or more[2, 3]).

More consistent evidence of mitral regurgitation is obtainable by means of the injection of Evans blue dye into the left ventricle during left heart catheterization.[4] In the presence of mitral regurgitation, immediate recovery of the medium from within the left atrium can be expected.

Left heart catheterization was performed in the prone position using the right paravertebral approach. Usually, right heart catheterization was done in a similar manner through a second needle.[2, 3]

The passage of a catheter into the left ventriele permitted measurement of any pressure gradients across the mitral and aortic valves. The recording of left atrial pulse pressure usually revealed the degree of mitral regurgitation accurately[2, 3] as long as the mean left atrium pressure was above 20 mm. Hg. At lower mean atrial pressures, the regurgitant pulse could frequently be evoked by exercise.

More recently, however, a simple dye injection technique has been more satisfactory for evaluating valve competency. In brief, the technique consists of injecting dye into the left ventricle via a catheter while aspirating from a separate left atrial needle. This is compared to a similar injection into the left atrium via the catheter while aspirating from the separate needle. As can be seen from Tables I and II, it is apparent that the test does not overestimate the degree of insufficiency.

The patients selected for surgical intervention were managed by the application of the Davila-Glover purse-string procedure as described by them, using the Davilla needle designed for this purpose.

The postoperative evaluation of results have been based upon the same triad which was employed in the preoperative selection of patients, namely, clinical evaluation, simultaneously combined right and left heart catheterization, and the injection of Evans blue dye into the left ventricle with subsequent recovery from the left atrium. An altogether too limited autopsy experience has also been included.

RESULTS

There were 13 hospital deaths (Table I). The majority of these (8) resulted from uncontrollable ventricular fibrillation at the time of surgery. Three patients died within 24 hours after completion of operation. One patient died 4 weeks after surgery as a result of subacute bacterial endocarditis due to hemolytic *Staphylococcus aureus* and the remaining patient died 2 months after operation of uncontrollable congestive heart failure. It has been apparent that this surgical procedure carries a very substantial risk for patients in the advanced categories which were selected. In an effort to overcome the problem of ventricular fibrillation at surgery, partial cardiac bypass was employed during the introduction of the purse-string suture in 3 patients. The first of these efforts appeared to be very successful and a patient having profoundly severe mitral insufficiency was thus operated upon with an excellent early clinical result. However, the signs of the regurgitation recurred in 6

TABLE I. DATA ON DEAD PATIENTS*

PATIENT	AGE	NYHA CLASS	CATHETERIZATION RESULTS								PREOPERATIVE DIAGNOSIS	SURGICAL OBSERVATIONS PURSE STRING		THERAPEUTIC RESULT
			RA	RV	PA	LA	LV	AO	BLUE DYE	V-C		PREOP.	POSTOP.	
H. R.	50	IV	(19)	72/12-16	72/38	(21)	108/18-20	108/66	N.D.	49	4+ M.I.	4+ M.I.	0 M.I.	Hospital death
E. G.	48	III	(7)	92/3-8		(8-13)	130/0-10	110/78	N.D.	24	4+ M.I.	4+ M.I.	2+ M.I.	Hospital death
I. F.	20	III		44/4-7	44/30	(12)	110/4-7	115/84	N.D.	6	2+ M.I.	4+ M.I. 1+ M.S.	0 M.I.	Hospital death
M. C.	25	IV		80/0		(12)	115/0-2	115/86	3+	10	3+ M.I.	4+ M.I.	0 M.I.	Hospital death
E. K.	35	IV				30/14		128/94	N.D.	8	3+ M.I.	4+ M.I.	0 M.I.	Hospital death
M. M.	23	III	(4)	25/2-4		(10)	100/	100/63	3+	3	3+ M.I.	4+ M.I.	0 M.I.	Hospital death
G. B.	33	IV	(17)	58/13-17		(22)	114/14-29	111/69	3+	5	3+ M.I.	4+ M.I.	0 M.I.	Hospital death
M. C.	34	IV	(10)	22/0-4		(8)	112/4-7	112/68	3+	4	3+ M.I.	3+ M.I.	0	Hospital death Subacute bact. endocarditis
F. K.	30	III				(9)	105/0-5	105/60	N.D.	14	4+ M.I.	4+ M.I.	2+ M.I.	Hospital death
J. M.	49	IV	(9)	45/6-9	45/20	(19)	100/5-11	100/56	N.D.	9	3+ M.I. Trace M.S.	4+ M.I.	2+ M.I.	Hospital death
†C. J.	40	III									M.S.	4+ M.I.		Hospital death
W. P.	18	IV	(13)			(18-35)	116/11-14	115/75	N.D.	10	4+ M.I.	4+ M.I. A.I.	0 M.I.	Hospital death
L. J.	42	IV	(14)	55/9-16		(38)			N.D.	6	2+ M.I.	4+ M.I.	0 M.I.	Hospital death
‡T. M.	25	III	Pre-op.	46/1	76/46	(26)	72/11	72/60	3+	6	3+ M.I.	3+ M.I., 2+ M.S.	1+ M.I., 1+ M.S.	Late death
		III	Post-op.		78/50	(20)	88/2-6	90/56	3+	0	3+ M.I.			
W. M.	26	III				(9)	120/7-10	120/60	3+	3	3+ M.I.	3+ M.I.	1+ M.I.	Late death
‡A. N.	32	IV	(15)	75/11-15		(24)	105/10-14	105/63	N. D.	21	4+ M.I.	4+ M.I.	1+ M.I.	Late death
J. B.	33	IV	(15)	58/11-18		(30)	93/0-4	90/63	3+	6	3 M.I.	3+ M.I.	1+ M.I.	Late death
B. D.	34	IV	(13)	103/7-11		(19)	103/5-8	103/83		9	3 M.I.	4+ M.I.	0 M.I.	Late death
§C. R.	23	III									M.S.	3+ M.I.	1+ M.I.	Late death

N. D. = Test not done.

*Composite of data on all patients who have died. Thirteen hospital deaths and 6 late deaths have been included.

†Not tested preoperatively.

‡Purse string suture cut-through.

§Incomplete left heart catheterization preoperatively.

months and the patient died 3 months later of congestive heart failure. Autopsy disclosed cut-through of the purse string (Fig. I). The remaining 2 patients operated upon with the assistance of partial cardiac bypass died of uncontrollable ventricular fibrillation during surgery.

There have been 6 additional deaths, each occurring after the patient was discharged from the hospital after having progressed through apparently successful surgery and uncomplicated convalescence (Table I). Three of these deaths were sudden and fairly early after surgery, each occurred at home, and autopsies were not obtained. The 3 remaining late deaths were preceded by evidence of recurrence of mitral insufficiency and somewhat prolonged congestive heart failure. Autopsy in these 3 patients disclosed evidence of cut-through of the purse-string suture.

The result of the application of the purse string as determined by the surgeon at the time of operation has been impressive. Indeed, in almost every instance, the palpable regurgitant jet was greatly reduced as measured by the tactile sense of the operator's intra-atrial finger. These observations have been in complete agreement with those published by Davila and Glover.

An observation of major importance was that the degree of mitral regurgitation varied markedly depending upon the work of the left ventricle. Whenever systemic arterial pressure was low, the improved cardiac output resulting from the initial tightening of the purse string raised the systemic pressure. As this happened, the tactile evidence of regurgitation increased, requiring further tightening of the suture. These observations would seem to be of importance in open heart surgery for mitral insufficiency, since visual estimation of the regurgitant flow, when the left ventricle is pumping very little blood, may be incorrect.

The disappointing experience of having the apical systolic murmur recur within a few days to a few weeks, or months, in most patients is one which cannot be forgotten. Among the survivors who were followed for a reasonable period of time, this event occurred in 7 of 11 patients. Following this auscultatory evidence of recurrence of mitral insufficiency, there ensued clinical evidence of loss of symptomatic gains.

In an effort to obtain objective information concerning the status of the survivors, postoperative left heart catheterization and blue dye injection was carried out on 10 of the 11 surviving patients (Table II). These tests were done more than 6 months after surgery and the data secured disclose that 7 patients now have mitral insufficiency of degrees equaling those demonstrated prior to operation. There are 4 patients in whom there remains no demonstrable regurgitation but 2 of these have evidence of mitral stenosis. A single patient who was retested 10 days after undergoing surgery was found to have had objective evidence of recurrence of severe valvular insufficiency. This has been suspected clinically as a result of recurrence of an apical systolic murmur 6 days after surgery. This patient has since died in chronic uncontrollable congestive failure and autopsy confirmed the recurrence of mitral regurgitation.

TABLE II. DATA ON LIVING PATIENTS*

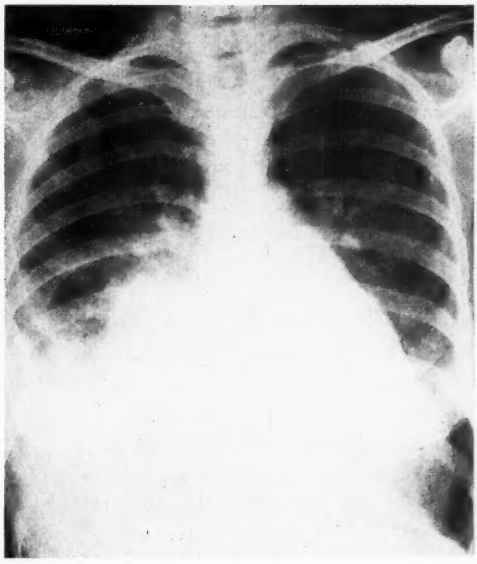
PATIENT	AGE	NYHA CLASS	CATHETERIZATION RESULTS								PREOP. DIAGNOSIS	SURGICAL OBSERVATIONS PURSE STRING		THERAPEUTIC RESULT
			RA	RV	PA	LA	LV	AO	BLUE DYE	V-C MM. HG		PREOP.	POSTOP.	
M. T.	40	III	(3)	26/4-5	26/15	(9)	109/2-4	109/72	2+	0	2+ M.I. 2+ M.S.	2+ M.I. 2+ M.S.	0 M.I. 1+ M.S.	Symptomatically improved
	42	II	(4)	26/2-4	26/16	(13)	140/6-8	140/107	1-2+	5	1-2+ M.I. 2+ M.S.			
M. B.	33	III	(8)	35/6-11		(13)	104/7-11	102/68	3+	0	3+ M.I.	4+ M.I.	1+ M.I.	Symptomatically unimproved
	34	III	(9)	40/1-8	39/19	(20)	120/2-9	111/79	3+					
D. C.	31	II	(10)	62/6-11		(24)			N.D.	6	3+ M.S. 2+ M.I.	3+ M.I. 2+ M.S.	0 M.I. 1+ M.S.	Symptomatically improved
	32	II	(4)	28/2-3		(10)	93/2-3	93/70	0	3	2+ M.S.			
C. B.	47	III				15-19	120/8-18	120/68	2+	2		3+ M.I.	0 M.I.	Symptomatically worse
	48	IV				(20)	135/7-10	135/74	3+	6	1+ M.S.			
C. A.	35	III	(10)	36/6-10		(18)	111/5-9	100/78	3+	3		3+ M.I.	0 M.I.	Symptomatically worse
	36	IV	(13)	45/7-12		(22)	109/4-13	112/92	3+	6				
W. M.	28	III	3-5	50/0-4		(25)	83/1-3	88/67	3+	5	3+ M.I. M.S.	4+ M.I. 1+ M.S.	0 M.I. 1+ M.S.	Symptomatically improved
	29	III	(7)	40/2-8		(22)	104/3-6	104/80	0	4	3+ M.S.			
M. F.	24	IV		38/8-10		(28)	88/6-8	76/10	3+	5	3+ M.I. 3+ M.S. 1+ A.S.	3+ M.I., M.S.	0 M.I. 1+ M.S.	Symptomatically improved
	25	II	(6)	32/1-6	28/15	(20)	107/0-6	105/84	3+	6	1+ M.S.			
L. J.	43	III	(9-14)	66/12	66/26	90/13	122/7-13	133/66	N.D.	50	4+ M.I.	4+ M.I.	0 M.I.	Symptomatically worse
	44	III	(9)	80/7-14	80/28	(24)		120/76	N.D.	35	4+ M.I.			
J. J.	40	III	(14)	73/12		(33)	114/0-18	115/85	2+	8		3+ M.I.	0 M.I.	Symptomatically improved
	41	III	(12)	85/6-12		(29)	165/10-14		0	0	4+ M.S.			
†C. K.											M.S.	3+ M.I. 1+ M.S.	1+ M.I.	Symptomatically unimproved
	34	II	(6)	49/5-13	49/28	(22)	106/2-10	88/74	0	2	3+ M.S. A.S.			
‡R. E.	27	III	(4)	28/0-2	19/11	(7)	128/4-8	107/60	3+	1	3+ M.I. A.S.	3+ M.I. A.S.	0 M.I.	Symptomatically unimproved

N. D. = Test not done.

*Composite of data on 11 surviving patients. The information includes that of pain before surgery as well as operative and postoperative findings.

†No preoperative catheterization.

N. D. = Test not done.
*Composite of data on 11 surviving patients. The information includes that of pain before surgery as well as operative and post-operative findings.
†No preoperative catheterization.



A.

B.

Fig. 1.—*A*, Chest film taken prior to surgery discloses large heart and right pleural effusion due to congestive failure. *B*, Photograph of heart made at autopsy 9 months after surgery. The purse-string suture can be seen to have cut through into left atrium just above mitral valve.

As further evidence of recurrence of valvular insufficiency we can present the autopsy findings of only 3 patients who have died 3 or more months after surgery. In 2 instances, the purse string had cut through the atrial wall and, in part, lay free in the atrial chamber just above the mitral leaflets (Fig. 1). In the third such instance, it was quite apparent that the purse string had cut through sufficiently to lose the effect although the suture had not entered the atrial cavity at any point.

SUMMARY

1. The circumferential purse-string suture described by Davila and Glover effectively controls mitral regurgitation at the time of application.

2. There has been a high incidence of recurrence of valvular incompetence, chiefly the result of cut-through of the purse-string suture.

3. A high mortality rate has been experienced.

4. There is evidence which indicates that we have produced a measureable degree of mitral stenosis in 2 patients both of whom have maintained control of a previous insufficiency.

5. Observations made during the surgery performed for mitral insufficiency indicate that the degree of valvular regurgitation may be underestimated under the conditions which prevail during open heart surgery.

6. The evaluation of surgical results of surgery for mitral insufficiency requires the utmost objectivity at our disposal. We feel that left heart catheterization studies, of the scope employed in this study, are mandatory.

REFERENCES

1. Davila, J. C., Glover, R. P., Trout, R. G., Mansure, F. S., Wood, N. E., Janton, O. H., and Iaia, B. D.: Circumferential Suture of the Mitral Ring, J. THORACIC SURG. **30**: 531-563, 1955.
2. Kent, E. M., Ford, W. B., Fisher, D. L., and Childs, T. B.: The Estimation of the Severity of Mitral Regurgitation, Ann. Surg. **141**: 47-52, 1955.
3. Fisher, D. L.: The Use of Pressure Recordings Obtained at Transthoracic Left Heart Catheterization in the Diagnosis of Valvular Heart Disease, J. THORACIC SURG. **30**: 379-396, 1955.
4. Fisher, D. L.: Catheterization of the Left Heart, *in* Zimmerman, H. A., editor, Intravascular Catheterization, Springfield, Ill., 1958, Chas. C Thomas, Publisher.

DISCUSSION

DR. J. C. DAVILA, Philadelphia, Pa.—Every year at this meeting someone invariably asks, "Are you still doing this operation?" and we have answered, "Yes, we are." I am of course very disappointed by the results which Dr. Kent, Dr. Neville, and Dr. Fisher have reported. This is a procedure which is technically exacting, and about which perhaps some details have not been fully emphasized or sufficiently clearly elucidated in our published reports. I would like to point out one feature which Dr. Neville and I discussed some days ago.

(slide) If the suture is placed in a manner which catches a band of epicardium in a loop and it is tied, a very disagreeable puckering is produced. Not only is this undesirable from the standpoint of hampering coronary arteries, but it is undesirable because the suture is unable to slide through after final tying to glide into the apex of the A-V

groove. This may appear to be a minor point but it is very important. In placing this portion of the suture it is necessary to come out with the suture and return through the same puncture in order that the suture lie entirely in fat. When it is tied it then glides into the proper position. This will eliminate to a very large degree, although not completely, the loosening of the suture. (slide) This is shown to point out the repetition of the loop back through the same orifice.

We have had only 1 case in which the suture was misplaced into the cardiac lumen; it occurred very early in our series. We have not had the suture cut through in any other patient or, in fact, in a large number of dogs in a period of 3 years of observation.

This slide simply shows the tracing of a patient whom Dr. Morrow catheterized in Washington, 1 month before surgery, 1 month after surgery, and 9 months after surgery.

(slide) This is the longest follow-up we have studied—a patient who was catheterized at 6 months and at 24 months postoperatively. You will note the left atrial pressure remains low, the pulmonary pressure remains low, the output doubles and remains that way, the pulmonary resistance is low. The heart size (by cardiothoracic ratio) remains small.

(slide) I wish to point out what has happened in our experience during the past 4 years. We have operated on 61 patients and I have divided them into three groups: 20 patients, 20 patients, and 21 patients, progressively. In the first 20 patients, there were six operative deaths, eight late deaths, and 6 are living; the next 20, six operative deaths, four late deaths, 10 are living. In the last 21, there were six operative deaths, one late death, and 14 are living. This represents not necessarily technical improvement but better selection of patients, and this is the crux in the success of this operation. It is not unlike the catastrophe which Dr. Potts referred to. When we operate on a patient in a far-advanced stage of disease, that patient is going to die whether we have corrected the insufficiency or not.

This is another aspect of the breakdown. Half of our patients roughly, 29, were preoperatively in medically intractable failure, 32 were in various stages of congestive failure but were not intractable preoperatively. Of the 29 in intractable states only 4 are living and only 2 of those are improved. Of the 32 in varying degrees of failure, 26 are living and 26 are improved.

We have not studied by recatheterization a sufficient number of patients completely to show comparable data before and after surgery, nor do we have time now to discuss that in more detail, but those we have studied have shown favorable results.

DR. LOUIS H. BOSHER, Richmond, Va.—My experience with this operative procedure is very limited, but so unsatisfactory that I wish also to support Dr. Kent's observation regarding it.

We have operated upon 6 patients, 2 of whom were operative deaths, that is, occurring early postoperative. There were 2 patients who died later, 1 six weeks and 1 five months later, and I would like to show two slides of the specimens.

(slide) You will see that in this patient the suture has cut through 5 weeks after surgery. There was clinical evidence of recurrence of mitral insufficiency. I might say that this suture was not enclosed in a pericardial tube as recommended by the authors of the procedure.

(slide) The second patient died 5 months later; the suture was enclosed in a plastic tube between the circumflex artery and the point of emergence in the transverse sinus. The suture did not cut through at this site but cut through elsewhere along the annulus. It is our opinion that if the annulus is markedly dilated, which is the situation in most of the patients, if you exert enough tension to eliminate the mitral insufficiency then almost certainly the suture will eventually cut through. We do have 2 patients alive who have had this procedure. They are somewhat improved. One of these patients exhibited evidence of elimination of mitral insufficiency for some months, but this has gradually returned and at the present time he has lost ground.

DR. J. NEVILLE (Closing).—In fairness to Dr. Davila, I would like to say that the technique which we used in placing the purse string was that originally described. It did not include the modification presented here. Whether this would influence the results which we have obtained I am not able to say at the present time.

We were quite impressed at the time of surgery by the ability of this operative procedure to control regurgitation. Despite our rather high operative mortality from ventricular fibrillation, we were rather reluctant to discontinue the procedure because it apparently offered some hope to a group of desperately sick patients.

An example of one of these patients is exemplified by this x-ray (slide). This patient required 3 months of hospitalization and intensive medical management to be brought into a reasonable cardiac compensation. Because of our prior experience with ventricular fibrillation in this type patient, she was operated upon using a pump-oxygenator by diverting only part of the cardiac output. With the blood continuing to return to the left atrium we were able to determine the degree of regurgitation more efficiently. The patient did survive the operative procedure despite a bout of fibrillation and a dramatic result ensued. Whereas previously she had been completely bedridden, within 3 months she was doing all of her housework. In addition, due to inadequate advice to the local physician, she was on a regular diet and off digitalis. Approximately 6 months after surgery, symptoms recurred and a progressive downhill course ensued. She was hospitalized and an autopsy fortunately was obtained.

I think this slide speaks for itself (purse string lying free in atrial cavity over one-half circumference).

There is one observation Dr. Kent made which I should like to amplify briefly. It was impressive at the time of placement of the purse-string suture to observe the relationship between the degree of regurgitation and the arterial blood pressure. Usually the blood pressure was quite low initially. As the purse string was tightened, the arterial blood pressure rose and the regurgitation would reappear or increase. Further tightening of the purse string would control the regurgitation but, as the arterial pressure rose further, the same phenomenon would take place. It was not until a normal systemic pressure had been obtained that we would be sure of the control of the regurgitation. I think this observation is pertinent to open operative correction of mitral regurgitation, for when the left ventricle is not completely filled, so that the intraventricular pressure does not reach normal levels, the degree of regurgitation may be very difficult to estimate. A false sense of security may result from merely the visual correction of the regurgitation.

TECHNICAL CONSIDERATIONS IN DECORTICATION FOR THE PLEURAL COMPLICATIONS OF PULMONARY TUBERCULOSIS

Paul C. Samson, M.D., Duane L. Merrill, M.D. (by invitation), David J. Dugan, M.D., Oakland, Calif., E. J. Shabart, M.D. (by invitation), Livermore, Calif., James Yee, M.D. (by invitation), Oakland, Calif., and Louis M. Barber, M.D. (by invitation), French Camp, Calif.

THE MODERN era of decortication for pleural complications in tuberculosis may be said properly to date from the end of World War II. During the war, the pathology of organizing hemothorax was finally settled. Many thoracic surgeons returned to civilian life with a considerable background experience in decortication for organizing hemothorax and post-traumatic empyema. In refocusing their attention on peacetime pursuits, it was only natural that renewed interest developed in treating the unexpandable lung in tuberculosis as well as in tuberculous empyema.

Another advance of the greatest importance likewise soon appeared: the development of specific chemotherapy for tuberculosis. As this discovery related to decortication, it meant that the prevention of invasive tuberculous infection made surgery safe in a contaminated field, thus enhancing the value of operative procedures already proved technically adequate.[1]

During the past decade a number of important contributory articles have appeared on decortication in tuberculosis.[2-11]

PATHOLOGY

While it was originally considered that the fibroblastic reaction in the pleural cavity (and the eventual development of a "peel") was in some way a specific response to the presence of blood, it became rapidly apparent that any insult to the pleural cavity would result in the same reaction. Air, pyogenic infection, specific effusions, viral effusions, chyle, and even neoplasm will elicit the same primary nonspecific response by the pleura: fibrin deposition on the exposed pleural surfaces; early fibroblastic and angioblastic proliferation; formation of a complete membranous envelope having both visceral and parietal components and containing air or liquid debris. Thick or thin, the fully developed membrane or peel in each case is composed of adult tissue relatively poor both in blood supply and nuclei.

From the Thoracic Surgical Services of Highland-Alameda County Hospital, Samuel Merritt Hospital, Peralta Hospital, Oakland, Calif.; The Veterans Administration Hospitals, Livermore and Oakland, Calif.; The San Joaquin General Hospital, French Camp, Calif.

Read at the Thirty-eighth Annual Meeting of The American Association for Thoracic Surgery at Boston, Mass., May 16-18, 1958.

The microscopic evidence of tuberculous infection is most frequently found along the inner or closed surface of the envelope away from the pleurae (Fig. 1). It is seldom seen invading the membrane or peel itself unless the tuberculosis is an extension outward from a pulmonary lesion. Therefore, the cleavage line between peel and pleura can nearly always be developed without disturbing the tuberculous infection within the envelope. In addition, adequate chemotherapy destroys the microscopic picture of specificity in an empyema in the majority of instances coincident with the disappearance of tubercle bacilli from the fluid (Fig. 2). This is a further and very important safety factor during and following surgery.

MATERIAL

During the 10-year period ending early in 1957, a total of 237 significant decortications were performed on the Thoracic Surgical Services of the indicated institutions. Nearly all of these were performed by the authors, except at Highland-Alameda County Hospital where at least half were done by former residents.

Of the 237 operations, decortication was the main procedure in 104, and an incidental but important adjunct to resection in 133.

Among the 104 patients, 39 operations were performed for unexpanded lung due to pneumothorax or pleural effusion; 6 for "false re-expansion"; 7 for pyogenic, 35 for pure tuberculosis, and 17 for mixed infection empyema. In this group, there were additional resections in 30 cases (4 of these with concomitant thoracoplasty) and *adjunctive* tailoring thoracoplasties (both concomitant and postdecortication in another 13 cases).

Among the 133 patients, no thoracoplasty was performed in 60 per cent; a preresection thoracoplasty was performed in 18 per cent, a concomitant thoracoplasty in 13 per cent, and a postresection thoracoplasty in 9 per cent.

INDICATIONS

The rationale of decortication in tuberculosis needs but brief comment. Obliteration of an intrapleural space, removal of infection, re-expansion of lung, and hemithoracic restitution with as near normal mobility as possible are the desiderata.

The indications are various: the unexpandable lung, following pneumothorax therapy or primary pleural effusion; the "false re-expansion"; intrapleural empyema, pyogenic, pure tuberculous, or mixed; and finally the incidental decortication necessary with many parenchymal resections for better re-expansion of remaining lung tissue.

There are, in addition, certain miscellaneous indications for decortication because of postresection complications. These arise rarely but are important in the individual case. Realization that a type of decortication is possible may induce the surgeon to embark on a highly successful salvage operation. In the case of occult fistula, for instance, a small empyema may be removed by extrapleural decortication of the empyema and re-amputation of the bronchial stump[12] (Fig. 3). A postpneumonectomy empyema has been likewise removed

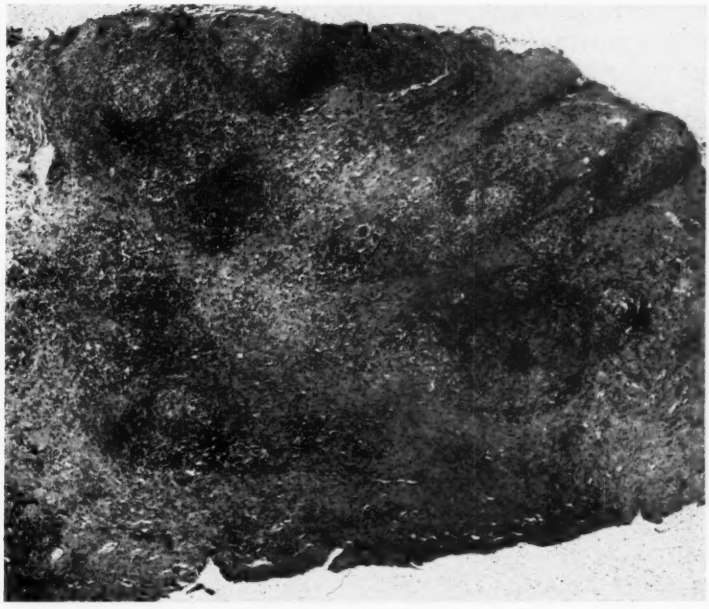


Fig. 1.—Pure tuberculous empyema. Photomicrograph of cross section of peel ×30. The tubercles are most numerous toward the inner surface of the envelope (above). The visceral juxtapleural edge (below) is free from tubercles.

Fig. 2.—Pure tuberculous empyema. Photomicrograph of cross section of peel ×30. showing inner surface of empyema cavity. This patient had prolonged chemotherapy prior to operation. One sees only a nonspecific inflammatory reaction. All evidence of specific tuberculous infection has now disappeared.

in toto. One patient in our series had three decortications because of air leaks and intrapleural infection following right upper lobectomy. The eventual result was satisfactory. Postresection intrapleural hemorrhage with organization is also a well-known indication.

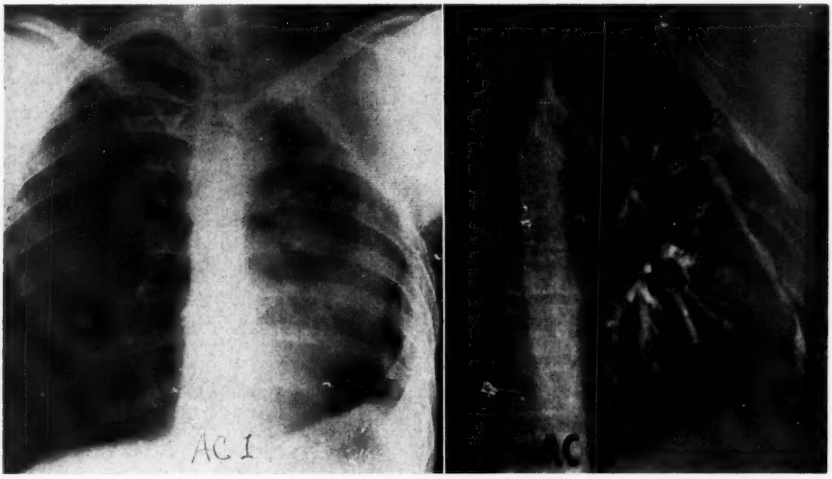


Fig. 3.—A patient with positive sputum which developed 4 years following left upper lobectomy and thoracoplasty performed elsewhere. The routine PA roentgenogram is unrevealing. The bronchogram demonstrates an occult fistula and empyema. Extrapleural exenteration of empyema with decortication and revision of the left upper lobe bronchial stump resulted in cure. (From Merrill, D. L., and Samson, P. C.: Am. Rev. Tuberc. 77: 561, 1958.)

PREOPERATIVE STUDIES

The final decision as to procedure depends upon the status of the underlying lung and this makes decortication in tuberculosis more complicated than when performed in other situations. It also means that frequently decortication may not be the sole operation, but is performed in conjunction with resection and/or thoracoplasty.

The status of the underlying lung must be studied in detail. Complete review of serial films, tomography, bronchoscopy, and bronchograms may all be indicated before a decision is reached. If the tuberculosis is inactive and originally not of great extent, it may be presumed that the lung can re-expand quite well. Decortication alone will then probably suffice. However, poor re-expansion may be expected in the presence of a high-grade stenosis of the mainstem bronchus and this may well be a contraindication to decortication. If there is active lobar disease or old pulmonary fibrosis, resection and/or thoracoplasty may be necessary in addition to decortication. Of course, a destroyed lung with pleural complications should be removed by the technique of pleuropneumonectomy.

Bronchograms are frequently valuable, but must be interpreted with care. In the absence of clinical symptoms, the widening and blunting of bronchi in

a collapsed lobe may not be anatomically permanent bronchiectasis but may be reversible on re-expansion following decortication (Fig. 4).

Fig. 4.—Pure tuberculous empyema. Pre- and postdecortication bronchograms showing reversal of "bronchiectasis" in left lower lobe. There were no clinical symptoms of bronchiectasis at any time. (From Merrill, D. L., and Samson, P. C.: Am. Rev. Tuberc. 77: 561, 1958.)

OVER-ALL SURGICAL CONCEPTS

The exact order of the concomitant operations most often applied in tuberculosis depends upon what is considered to be the main pathologic disorder. In cases of empyema, or unexpandable lung, decortication is the primary operation and the first procedure undertaken. On the other hand, resection of some type may be proceeded with first and decortication of varying degree then acts as a supplemental operation. Usually the decision concerning a concomitant thoracoplasty is delayed until it is seen whether pulmonary re-expansion is adequate following decortication and/or resection (see Fig. 16). In some instances the tailoring thoracoplasty has been a subsequent operation when re-expansion was not satisfactory (see Figs. 17-18). Rarely, a preliminary thoracoplasty (as a separate operation) has been undertaken when judgment indicated with fair certainty that the lung would inadequately fill the hemithorax.

Postoperative pneumoperitoneum can likewise help in minor space problems. I believe this has been used with increased frequency in the past 2 or 3 years.

Certain surgical principles have evolved in our clinics which apply to all types of decortication. Nonflammable anesthesia is used. This allows the employment of the electrocoagulation which is a great timesaver in many instances in which multiple areas of punctate bleeding are present. The standard approach is posterolateral, removing the fifth or sixth rib. We prefer to remove a rib because we believe that a more secure chest wall closure can be performed. In general, fine nonabsorbable suture material is used throughout. If raw lung

tissue needs to be repaired, we employ fine catgut on an atraumatic needle. At least two right-angle drainage tubes of from 36 to 40 Fr. are used, one anteriorly in the second interspace and one posterolaterally in the eighth interspace. Occasionally a third and posterior tube is used, particularly when the superior segment of the lower lobe has been removed. If numerous small lung leaks persist, some type of suction must be employed and we have become accustomed to the use of high-vacuum, high-flow suction drainage with a negative pressure available, of from 25 to 50 cm. of water. Admittedly, there is continuing discussion that high, negative intrapleural pressures may help maintain fistulas. It is our present belief that this may well be true of *bronchial* fistula, but not of multiple small air leaks. High-vacuum suction seems the best means of obtaining prompt pulmonary re-expansion under the latter circumstances. Roe[13] has recently considered these matters at length.

General standards have been set up in caring for the chest wall in cases of prior drainage. Sinuses, including pleurocutaneous flap drainages of the Eloesser type, are excised down to the ribs, dissected loose, and turned in so they may be removed by the later intrathoracic operation. The chest wall is then closed in layers over these areas, instruments, gowns, and gloves changed, and the main thoracotomy incision made under sterile conditions. In the case of tube tracks which are expected to be in the proper place for drainage following operation, these may likewise be excised to remove scar tissue. However, we have not disturbed the more recent ones and have used the same drainage sites following decortication.

Recently, hypothermia has been used on several patients whose vital capacity was extremely low and when it was hoped decortication would improve ventilatory reserve (see Figs. 12-13). Within the past few months, Brewer and King[14] have independently reported on a similar use of this modality.

Proper antituberculosis drug coverage is of the greatest importance when the pleural and/or pulmonary disease is or has been infectious. If the organisms are sensitive, or if no tubercle bacilli are found, the standard drugs are continued through surgery and for many months thereafter. Streptomycin is placed in the pleural cavity only when there has been gross contamination, but we must confess that we have no statistical justification for its use intrapleurally. When the tubercle bacilli are resistant to streptomycin, PAS or INH, we consider it mandatory to use other drugs. Pyrazinamide, viomycin, or cycloserine are generally preferred. The drug of choice is commenced some 14 days before operation and continued from 4 to 6 weeks postoperatively. In mixed infection empyema, sensitivity studies are performed on the pyogenic organisms present and the appropriate antibiotic is then added to the regimen and continued for from 7 to 10 days following surgery.

THE UNEXPANDABLE LUNG-PNEUMOTHORAX COMPLICATIONS

The nonexpanded lung due to pneumothorax, or primary pleural effusion, represents a double hazard to the patient: there is loss of pulmonary function which is at least partly restored by decortication; persistence of a space invites infection sooner or later.

Decortication under these circumstances is a simple, effective, and highly safe procedure with excellent results. Using the standard approach, the parietal peel is incised and the center of the envelope entered directly. Standard visceral pleural decortication is carried out under intermittent positive pressure by the anesthetist. Depending upon the size of the pocket, its duration, and a previous knowledge of whether the chest wall is fixed, we may or may not do a parietal pleural decortication. It is not routine as it increases the operating time and the bleeding. If the pocket is a lateral one, it is well to separate all areas of adherence between visceral and parietal pleura down to the diaphragm and to the hilum so that circumferential expansion can take place. In most cases of this type, there has been no need for either resection or thoracoplasty (Fig. 5). Rarely, the discovery of a fibrotic, poorly expansile lobe has led us to perform a tailoring concomitant thoracoplasty.

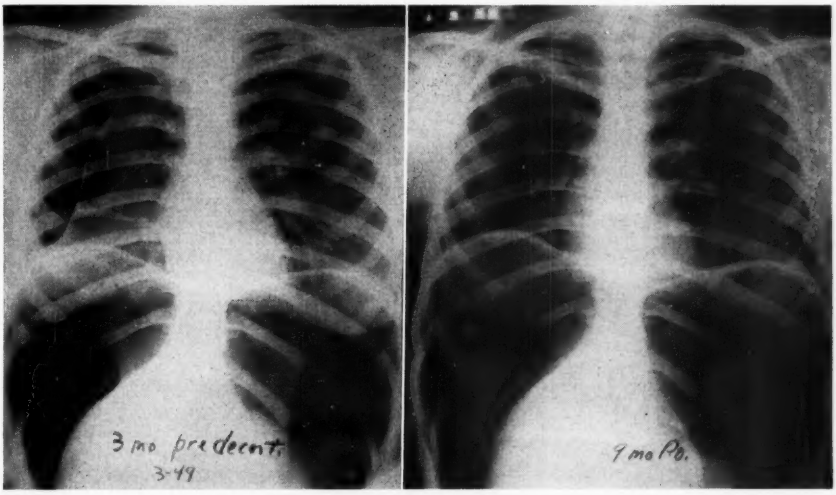


Fig. 5.—Excellent result from decortication for unexpandable lung following pneumothorax therapy of 5 years' duration.

Granted that few pneumothoraces are induced these days, we should nevertheless include a word on the management of the complicated ineffectual pneumothorax. In spite of the most careful management and early abandonment of an ineffective pneumothorax, there may be prompt fluid formation. By implication, the pulmonary disease is almost always active. If the lung cannot be immediately re-expanded by aspiration, early surgery is indicated. In general, thoracotomy with evacuation of fluid, decortication of the normal lung, and possibly lobectomy should give a more satisfactory result, although surgical drainage of an *infected* pleural cavity may be the preferable alternative.

If lobectomy is indicated as part of the procedure but, bronchoscopically, the bronchus is not considered suitable for transection, a temporizing extrapleural thoracoplasty may be instituted to help control parenchymal disease as well as obliterate the pleural cavity. Occasionally, a *technically adequate*

pneumothorax is ineffective in closing a lobar cavity. Re-expansion is not successful because a fibrous membrane has developed. In this situation, we favor decortication, lobectomy, and possibly concomitant thoracoplasty, or postoperative pneumoperitoneum. Here, decortication is essential for adequate re-expansion of the remaining lobe (Fig. 6).

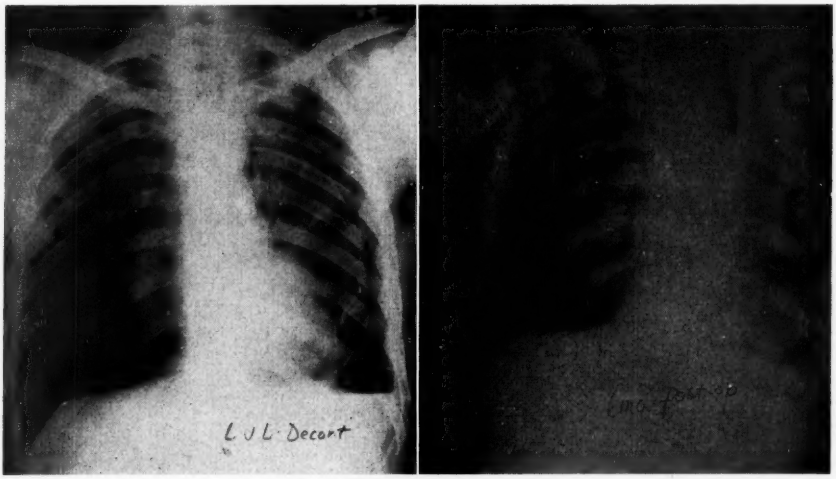


Fig. 6.—A technically adequate pneumothorax, but there is a residual cavity in the left upper lobe and beginning fluid formation. Postoperative roentgenogram following decortication, left upper lobectomy, and concomitant thoracoplasty (ribs 2, 3, 4).

THE "FALSE RE-EXPANSION"

This condition usually has been a complication of pneumothorax or primary pleural effusion. Obliteration of the pleural space by mediastinal and cardiac shift rather than by effective pulmonary re-expansion has resulted in continued disability because of tension phenomena affecting the heart, thoracic parietes, or diaphragm. A calcium plaque may rarely develop. The patients in this group have been few but a greater number may well be symptomatic. We suggest that a critical review of patients whose pneumothorax has been abandoned might turn up an appreciable number of candidates for operation. These patients present a varying symptomatology. Frequently the symptoms are not crippling but very annoying. Persisting thoracic discomfort or actual pain, made worse by exertion; dyspnea and breathlessness; pressure sensations in the chest; chronic fatigue; recurrent formation of fluid; palpitation and tachycardia; and "asthma" have all been recorded. In 1 patient, a systolic heart murmur became progressively louder apparently due to cardiac torsion (Figs. 7 and 8). On examination, these patients have flattened chests on the affected side. The intercostal spaces are narrowed and retracted, and limited inspiratory expansion is quite obvious (Fig. 9). In the few patients, decortication, where employed, has been highly successful; Mulvihill[3] and others have likewise operated in similar situations to improve pulmonary function and thus allow for needed surgery on contralateral active lesions.

At operation, the peel will be double layered and, in general, the two layers will be adherent. They may, however, be intermittently separated by small lacunae of fluid or by coalescent calcium deposits (Fig. 10). The latter are always on the inside of the envelope and parietal pleurectomy may well be advisable when dealing with calcium so that the whole plaque may be removed in

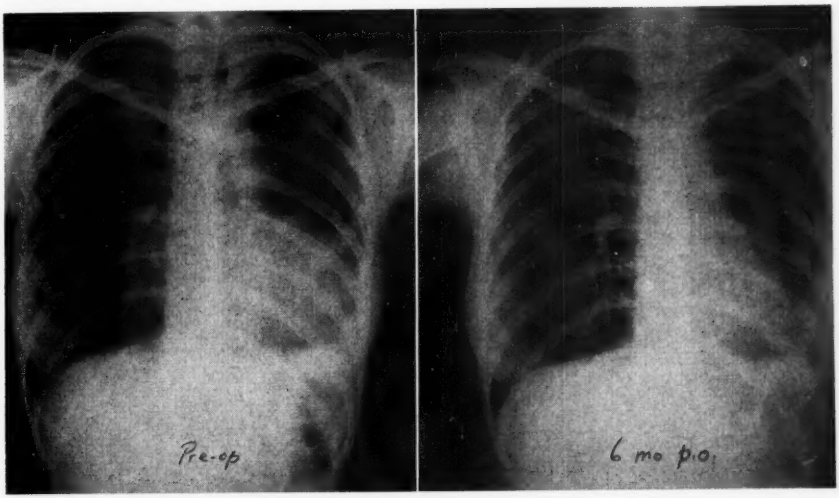


Fig. 7.—"False re-expansion" following 5 years of pneumothorax therapy. Complaints of tightness in chest, and fatigue. Progression of systolic cardiac murmur from Grade I to Grade IV with re-expansion. Following decortication, loss of symptoms and marked regression of murmur.

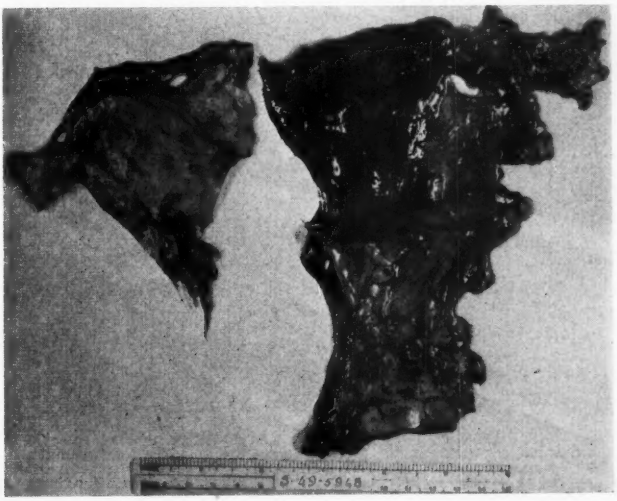


Fig. 8.—Peel from patient shown in Fig. 7. There is demonstrated the reflection from visceral to parietal and diaphragmatic surfaces. The fibrous tissue composing the peel was rigid and relatively avascular.

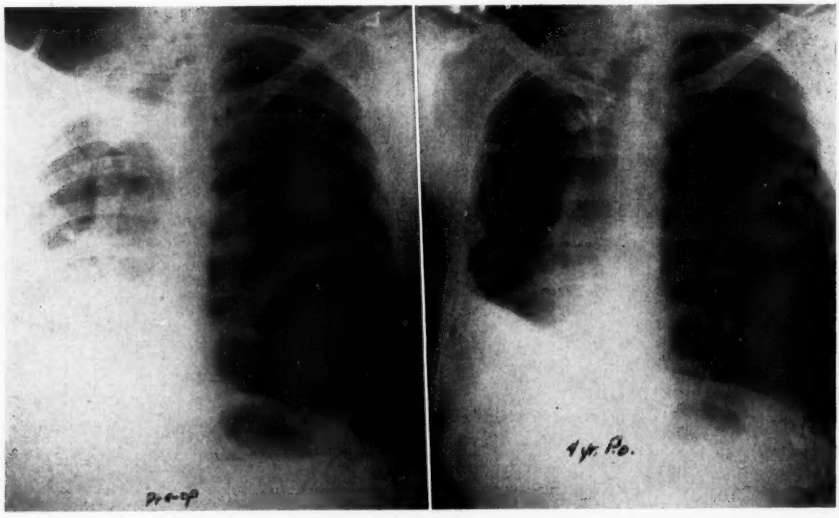


Fig. 9.—Inactive tuberculosis. False re-expansion following 16 years of pneumothorax therapy. Decortication. A poor radiologic result. Clinically, patient has lost all preoperative symptoms of dyspnea, thoracic pressure, tachycardia, and frequent colds.

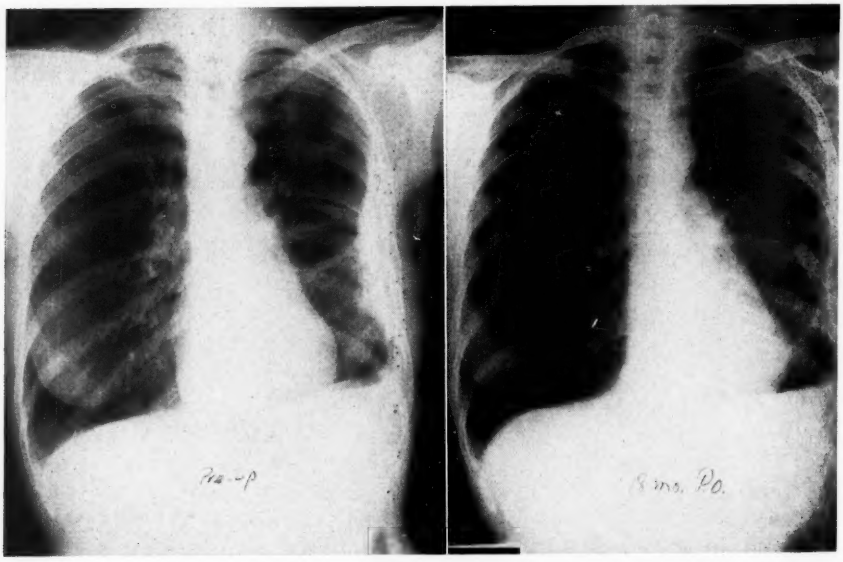


Fig. 10.—A woman, aged 48, with presenting complaints of thoracic discomfort, weakness, severe cough, intermittent "asthma," and intermittent fever. Artificial pneumothorax complicated by fluid 30 years before. "False re-expansion." Extrapleural exenteration and decortication of sac containing large calcium plaque. The scanty fluid in the envelope contained viable tubercle bacilli. Today she is without symptoms.

toto rather than piecemeal. En masse removal is much to be preferred since it is almost impossible entirely to remove the calcium bit by bit. Remaining pieces act as foreign bodies and infectious complications are certainly more frequent under these circumstances.

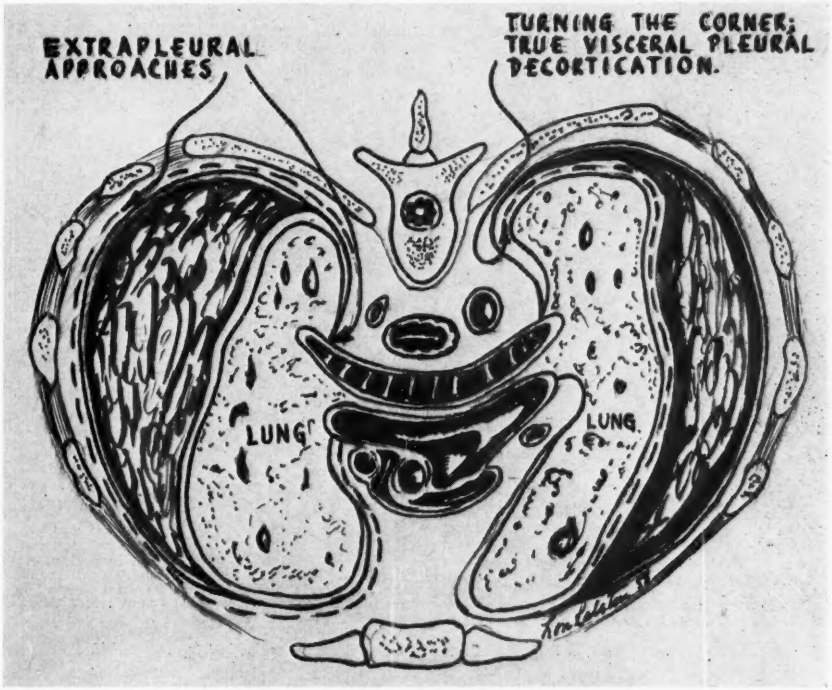


Fig. 11.—Diagrammatic sketch of surgical approaches. On the left is demonstrated the principle of extraparietal pleural dissection for the preliminary freeing of an empyema sac, or the direct approach to the hilum for pleuropneumonectomy. On the right is demonstraied a change from extrapleural to interpleural dissection by incision of the parietal pleura "at the corner." True visceral pleural decortication can then be undertaken and the envelope removed en masse without spillage.

EMPYEMA

Decortication has made the greatest difference in the treatment of empyema.

In acute pyogenic empyema, it has generally been considered proper to perform a preliminary surgical drainage. The empyema might well become obliterated, but this has been rare in our experience when the pyogenic infection was a complication of an established pneumothorax. Decortication would then be necessary after the empyema had become stabilized (Figs. 12 and 13). In selected instances, we believe that primary thoracotomy and decortication without preliminary drainage is a logical procedure.

In chronic empyema from whatever source, one has but to remember the deforming extrapleural and Schede thoracoplasties of yesteryear to realize how much better off are these patients today. The surgical management of chronic

empyema in tuberculosis embodies the principle of attempting *to remove the entire empyema sac or envelope without opening or spilling the contents.* It is surprising how often this can be accomplished even with an empyema of 20 years' duration. This basic technique as stressed by Weinberg[4] and others was first independently employed by us in mid-1947 (Fig. 11).

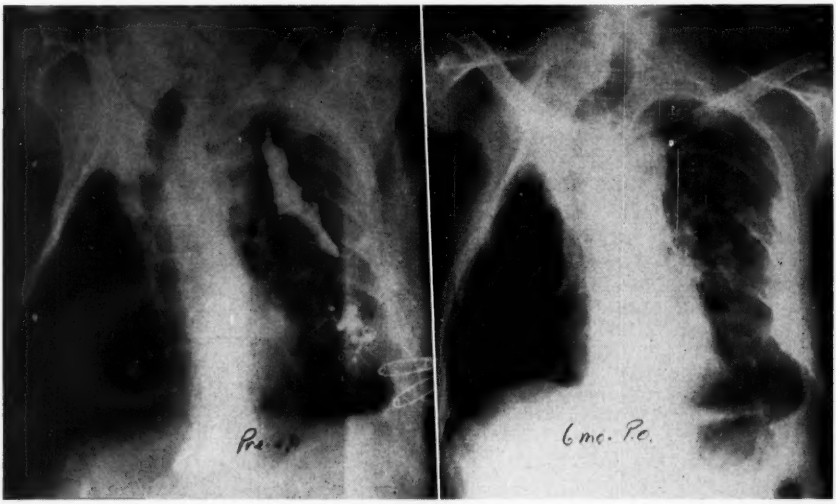


Fig. 12.—A 43-year-old man with inactive tuberculosis. Right bilobectomy and concomitant thoracoplasty 4 years before. Left pneumothorax complicated by pyogenic infection previously drained. Patient dyspneic on slightest exertion. Resting pulse frequently over 100. Left chest fixed. Under *hypothermia,* extrapleural exenteration of empyema and decortication. During the 5-hour operation, the pulse averaged 70. At present, the patient's left thorax is movable and without draining sinuses. He is not dyspneic on moderate exertion.

Fig. 13.—Cross section of peel from patient demonstrated in Fig. 12. The parietal segment (below) was mobilized extrapleurally. The "corner" (to the right) was turned by incising the parietal pleura and developing a line of cleavage between visceral pleura and peel (above).

On the chest wall side, the parietal pleura is removed with the peel most of the time, a fact not generally appreciated. Actually, we do parietal pleurectomy from preference because a cleavage plane is far more easily developed extrapleurally than it is between pleura and peel; likewise there is much less chance of breaking into the envelope. With the outer portion of the empyema sac completely freed from the chest wall, there is sometimes difficulty because of

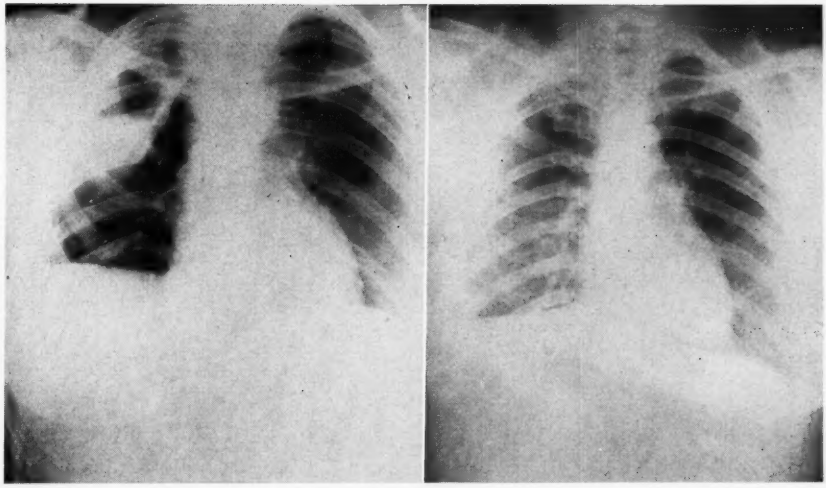


Fig. 14.—Chronic tuberculous empyema of 4 years' duration. This was the earliest of our en bloc exenterations of an empyema sac. Performed April 4, 1947. There was no chemotherapy employed.

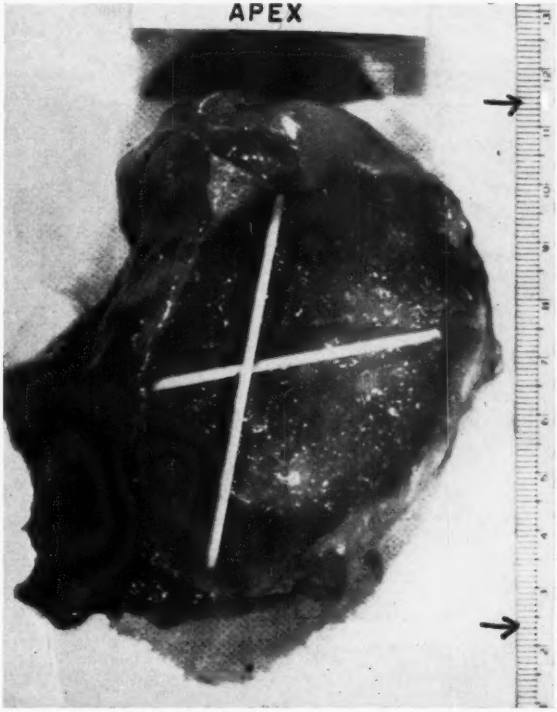


Fig. 15.—Opened mediastinal aspect of empyema sac from patient shown in Fig. 14, showing the rough and granular appearance of the inner surface of the envelope. Microscopically, this peel showed extensive tuberculous infection.

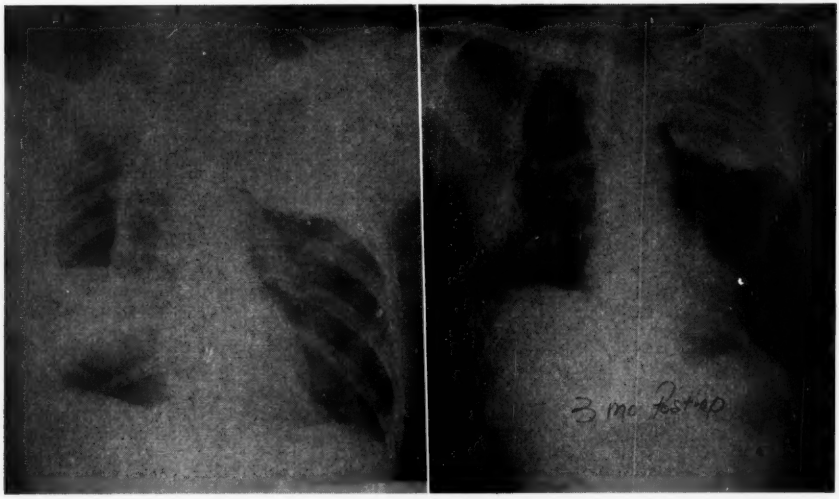


Fig. 16.—Far-advanced inactive tuberculosis with fibrotic right upper lobe. Left extrapleural oleothorax. Right mixed infection empyema: pneumothorax, 15 years; tuberculous empyema, 13 years; pyogenic invasion, 3 months, controlled by aspiration and antibiotic injection. Total en bloc removal of empyema sac without spillage, and concomitant thoracoplasty, ribs 2-6. Patient now employed full time as a schoolteacher.

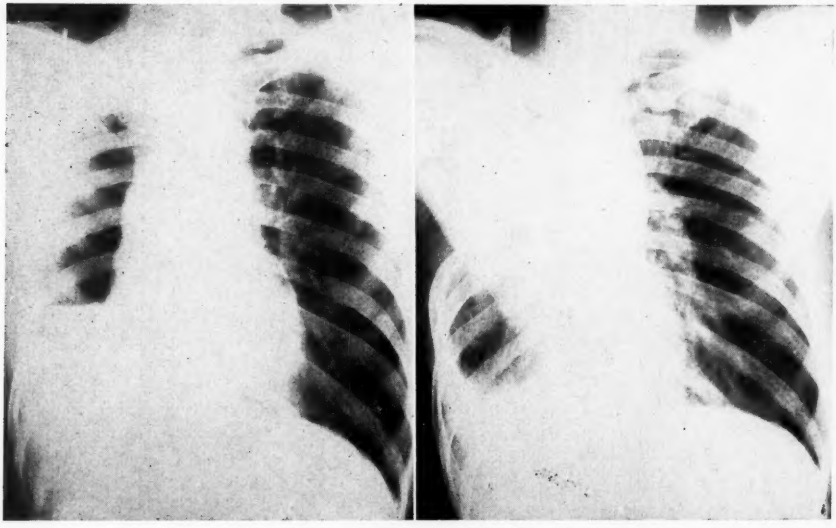


Fig. 17.—Pure tuberculous empyema of 5 years' duration. Fibrosis of right upper lobe. Extrapleural removal of empyema sac with visceral decortication was followed by poor expansion. A secondary thoracoplasty was successful in obliterating the apical pocket.

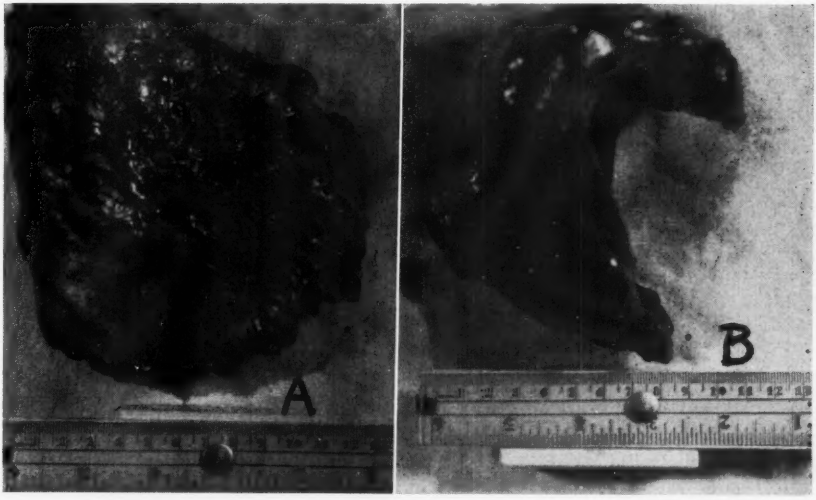


Fig. 18.—Two views of empyema envelope from patient shown in Fig. 17. *A*, External or parietal surface. The diagonal rib markings are characteristic. *B*, Apical aspect. The two edges (or corners) where the dissection changes from extrapleural to interpleural are particularly well demonstrated.

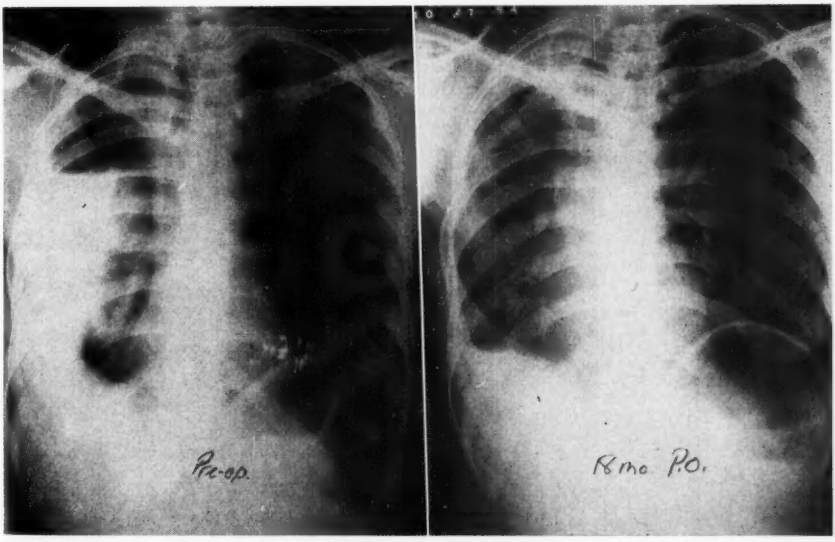


Fig. 19.—A 39-year-old woman with inactive tuberculosis. Pneumothorax of nearly 12 years' duration and pure tuberculous empyema for an additional 3 years. Vital capacity, 1,500 c.c. Maximum breathing capacity, 58.8 L. per min. Extrapleural mobilization of empyema sac and removal without spillage by visceral pleural decortication (operation by Dr. K. L. Hardy). An excellent result. There was a 25 per cent improvement in ventilation studies.

fullness of the sac. A bit of technique sometimes forgotten is to plunge a large-bore needle with suction attached into the empyema cavity and remove fluid and/or air to the greatest extent possible. This will collapse the sac and thereafter there will be plenty of room to work. The critical point of the operation employing pleurectomy is recognition of the exact line where the two pleurae

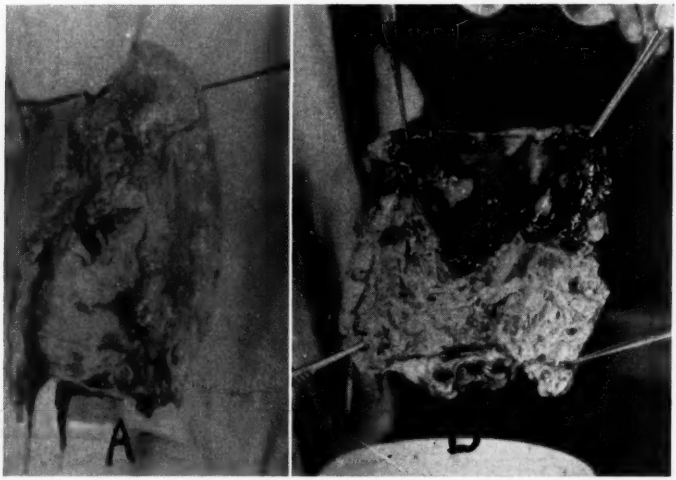


Fig. 20.—The surgical specimen from patient shown in Fig. 19. *A*, Mediastinal (visceral pleural) aspect of empyema sac. *B*, Interior of sac showing shaggy fibrin and amorphous debris.

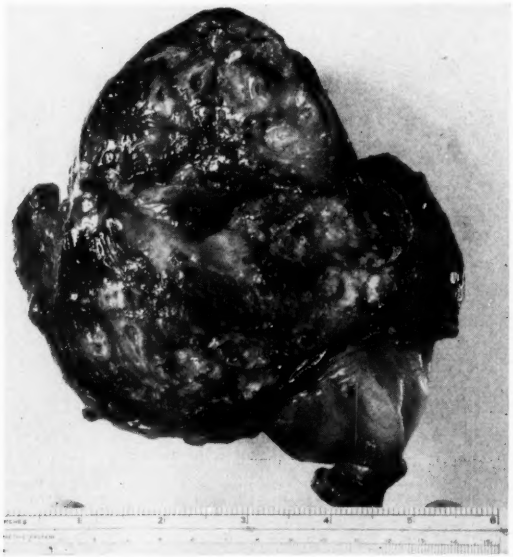


Fig. 21.—Destroyed lung and empyema sac removed together en masse by extrapleural approach and pleuropneumonectomy.

themselves come into apposition. When this point is reached, further extrapleural dissection is unnecessary and unwise. The cleavage plane should then be changed from extrapleural to interpleural and the hilar structures identified and freed. One can then come back on the visceral pleural surface and, by degrees, complete a true decortication. When the technique is successful, a bag

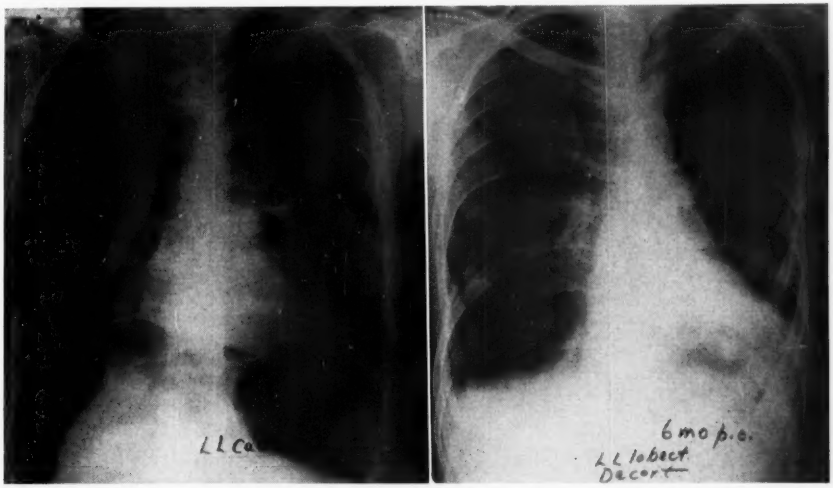


Fig. 22.—Pre- and postoperative roentgenograms of a patient who had left lower lobectomy and incidental decortication of the left upper lobe. The preoperative film gives no hin as to the extent of the peel.

Fig. 23.—The peel from patient shown in Fig. 22. It is 2 mm. in thickness, firm and inelastic. Satisfactory re-expansion of the left upper lobe would have been impossible without adequate decortication.

filled with pus may be totally removed from the thoracic cavity without leakage of contents. The advantages, particularly, in tuberculous infections, are obvious (Figs. 12-20). A natural extension of this means of treating empyema is the technique of Sarot[15] in which the extrapleural separation is carried to the hilum and pneumonectomy performed. Thus, a destroyed lung and an empyema can be completely exenterated en masse without spilling (Fig. 21).

INCIDENTAL DECORTICATION ASSOCIATED WITH RESECTION

The incidental or casual decortication which accompanies many resections deserves to be stressed. We found that this was significant enough to be described in approximately one third of our resections for tuberculosis in the series studied. In these, due either to a former pneumothorax, dry pleurisy, or what not, there had developed a thin, translucent, but inelastic glove over the

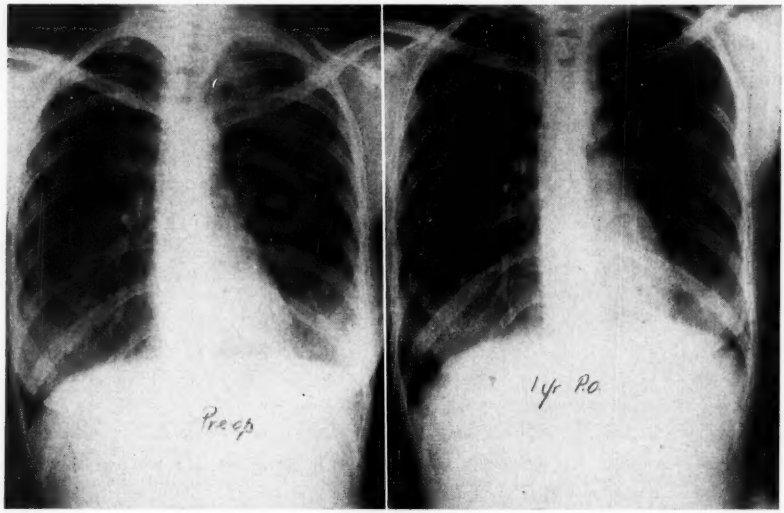


Fig. 24.—Reappearance of tuberculous cavity following abandonment of technically adequate pneumothorax. Apical posterior segmentectomy, left upper lobe, and incidental decortication of remaining lung. Note re-establishment of left costophrenic sulcus, postoperatively.

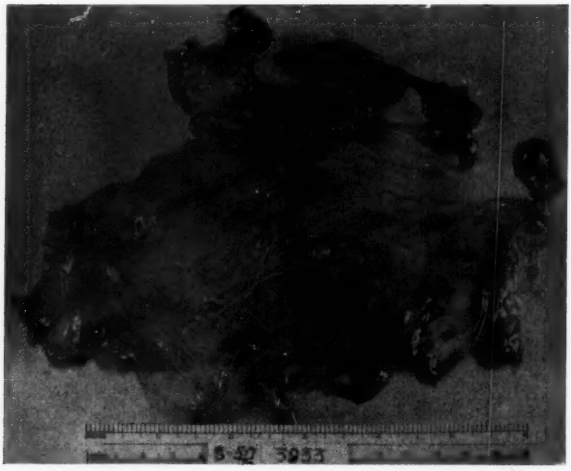


Fig. 25.—The decorticate removed at operation on patient shown in Fig. 24. The peel is semitransparent and edematous, but completely inelastic.

remaining lobes. Removal was thought necessary since the residual lung tissue would expand poorly and the success of the resection would become jeopardized thereby.

The technique of the casual decortication is not difficult. The important point is to recognize that a thin, often translucent peel is present and that such peel is relatively inelastic. It is amazing how tough-fibered such peels are and it is highly gratifying to note the improvement of re-expansion following their removal. The proper cleavage plane is identified after meticulous incision of the peel with the lungs supported by moderate positive pressure. Small blunt gauze or cotton dissectors will disclose a glistening visceral pleura covered with minute punctate hemorrhages from the divided vessels entering the peel. If an air leak develops, the visceral pleura is being dissected with the peel. This peel can be removed in strips and sometimes in fairly large flattened pieces corresponding to the entire external surface of a lobe. Fissures should be redeveloped and any tendency to infolding be corrected. In general, we have carefully dissected around any evidence of old subpleural disease, leaving the peel attached to these areas. Whenever any appreciable air leak has been encountered, it has been repaired, often using bits of the peel to oversew these areas (Figs. 22-25).

RESULTS

The results of operation have been quite satisfactory. In the group of 104 patients whose decortication was the main operation, there were 4 deaths (3.8 per cent). A review of postoperative findings reveals 79 cases (77 per cent) were graded Good to Excellent. This means prompt pulmonary re-expansion with little or no haziness; a clear or slightly blunted cardiocostophrenic sulcus; adequate motion of diaphragm and thoracic cage, and a satisfactory improvement in pulmonary function studies in cases where these were made.

Twenty-one cases (19 per cent) were graded as a Fair to Poor result. One cause was poor pulmonary re-expansion, usually associated with persisting pleural reaction, mediastinal shift, and elevated diaphragm (see Figs. 9, 12, 13). In nearly every instance, the basic reason seemed to be prior extensive disease with fibrosis of the lung. Nearly all of these patients, however, were greatly improved clinically in spite of the poor *radiologic result,* or failure to demonstrate improvement in pulmonary function. They lost the sense of pressure and tightness in their chests, were relieved of frequent aspirations, or became rid of a source of chronic infection.

A second cause for a "Poor Result" was the persistence of pleural space infections with residual sinuses in the thoracic wall. All of these eventually responded to adequate drainage or secondary local plastic procedures. Two patients had a spread of the pulmonary tuberculosis. Both responded to antituberculous therapy and today are well.

In the group of 133 patients whose decortication was incidental to resection, there were 3 deaths (2.3 per cent). Good to excellent results were

recorded in 104 cases (78 per cent). The complications in 26 cases (19.7 per cent) were considered primarily the result of the resection rather than of the decortication.

Unfortunately, we cannot present a large number of pre- and postoperative pulmonary function studies. In the past,[16] a fear has been expressed that some poorly expansible lungs might actually be a detriment to the patient, postdecortication, because of decreased ability to oxygenate blood. We have seen no patient in which this became clinically evident. It is our belief that living aerating lung, even though poor functionally, is a better "filler" for the hemithorax than are inert plastics or organizing blood and serum.

SUMMARY

1. A study has been made of 237 patients undergoing decortication during the past 10 years. In 104 of these, decortication was the main operation; in 133, it was incidental but an important adjunct to the more major resection. The over-all mortality was low (2.9 per cent) and the results generally satisfactorily.

2. Relief of thoracic discomfort and dyspnea, removal of sources of actual or potential infection, and adequate hemithoracic restitution were recorded in 77 per cent.

3. Decortication has been indicated in the following general classifications: the unexpandable lung due to long-term pneumothorax therapy, or primary pleural effusion; the "false re-expansion"; pyogenic, pure tuberculous, and mixed infection empyema; decortication incident to resection.

4. The technical details of decortication in the various categories have been described.

5. It has been stressed that frequently decortication may necessarily be combined with pulmonary resection and/or thoracoplasty depending upon the status of the underlying lung (bronchial stenosis, fibrosis, active tuberculous infection, clinically evident bronchiectasis).

6. Properly employed, decortication with or without parietal pleurectomy has proved itself a safe and effective procedure in treating the pleural complications of pulmonary tuberculosis.

REFERENCES

1. Samson, P. C.: The Prophylactic Administration of Streptomycin Before and After Major Thoracic Surgical Operations, Am. Rev. Tuberc. **58:** 38-41, 1948.
2. Gurd, F. B.: Decortication in Chronic Empyema of Tuberculous Origin, J. THORACIC SURG. **16:** 587-601, 1947.
3. Mulvihill, D. A., and Klopstock, R.: Decortication of the Nonexpandable Postpneumothorax Tuberculous Lung, J. THORACIC SURG. **17:** 723-742, 1948.
4. Weinberg, J. A., and Davis, J. D.: Decortication of the Unexpanded Tuberculous Lung Following Pneumothorax, J. THORACIC SURG. **18:** 363-371, 1949.
5. Gordon, J., and Welles, E. S.: Decortication in Pulmonary Tuberculosis Including Studies of Respiratory Physiology, J. THORACIC SURG. **18:** 337-362, 1949.
6. Samson, P. C.: Decortication in Pulmonary Tuberculosis, Tuberculology **12:** 142-149, 1951.
7. Thomas, G. I., and Jarvis, F. J.: Decortication in Primary Tuberculous Pleuritis and Empyema With a Study of Functional Recovery, J. THORACIC SURG. **32:** 178-189, 1956.

8. Samson, P. C.: Some Surgical Considerations in Pulmonary Decortication, Am. J. Surg. **89**: 364-371, 1955.
9. Waterman, D. H., Domm, S. E., and Rogers, W. K.: A Clinical Evaluation of Decortication, J. THORACIC SURG. **33**: 1-20, 1957.
10. Langston, H. T., and Correll, N. O., Jr.: The Surgical Treatment of Pleural Tuberculosis. The John Alexander Monograph Series, No. I. The Surgical Management of Pulmonary Tuberculosis, Springfield, 1957, Charles C Thomas, chap. VIII.
11. O'Rourke, P. V.: Decortication in the Treatment of Pleural and Pulmonary Tuberculosis. The John Alexander Monograph Series, No. I. The Surgical Management of Pulmonary Tuberculosis, Springfield, 1957, Charles C Thomas, chap. IX.
12. Merrill, D. L., and Samson, P. C.: Bronchography in Pulmonary Tuberculosis, Studies on Its Value in Patients Being Considered for Surgery, Am. Rev. Tuberc. **77**: 561-593, 1958.
13. Roe, B. B.: Obliteration of the Pleural Space Following Pulmonary Resection, Utilizing High-Flow, High-Vacuum Suction. (Presented at the 1958 Meeting of the Pacific Coast Surgical Association), Am. J. Surg. To be published.
14. Brewer, L. A., and King, E. L.: Hypothermia in Thoracic and Thoracoabdominal Surgery. (Presented at the 1958 Meeting of the Pacific Coast Surgical Association), Am. J. Surg. To be published.
15. Sarot, I. A.: Extrapleural Pneumonectomy and Pleurectomy in Pulmonary Tuberculosis, Thorax **4**: 173-223, 1949.
16. Wright, G. W., Yee, L. B., Filley, G. F., and Stranahan, A.: Physiologic Observations Concerning Decortication of the Lung, J. THORACIC SURG. **18**: 372-388, 1949.

DISCUSSION

DR. JOSEPH GORDON, Albuquerque, N. M.—I should like to carry on the discussion which you have just heard regarding the thin membranous peel reported in some types of cases. In decortication done for re-expansion, if the cortical peel is not carried down to the visceral pleural surface this thin membranous material, that looks almost exactly like visceral pleura, may be left behind, and the re-expansion is not nearly so good. When this membranous peel is removed, unfolding of the lung occurs, yielding maximum inflation. I should like to show some slides of this thin membranous material.

Dr. Samson showed in his second slide the thick visceral cortical peel removed, and evident in only one area was a portion of the small, thin membranous material.

(slide) In this particular slide, we show the dense cortical peel and the thin membrane which you can readily see.

(slide) In this next slide is shown the thin membrane that is removed separately, showing a moderate amount of elastica.

(slide) On further examination of this peel, in the next slide, we see these pleural clefts, large ovoid space in which there is a group of cells which are mesenchymal in origin. This is the thing which in my opinion is responsible for the development of an unexpandable lung. The details of the method by which the lung remains unexpanded have not been thoroughly explained. The primary development of this membrane over the collapsed lung is apparently the cause.

(slide) One can see another of the clear spaces with the small blood vessels contained therein. These mesenchymal cells therefore are not arising from the blood vessel itself but are a separate entity.

There apparently is some trigger mechanism which is not yet clear and which, when the lung is under collapse, apparently causes this membranous material to develop. This thin membrane, not unlike the visceral pleura, holds the lung down so that it cannot re-expand. From this point onward it is easy to explain why we have the development of a dense cortical peel. The fibrin organizes into fibrous tissue and the lung is obliged to remain down. But, in order for this to occur, it is necessary for something to prevent the lung from fully re-expanding as it normally would. The membranous development may happen whether it is a hemothorax, whether it is a pyogenic empyema, whether it is long-standing pneumothorax with the presence or absence of fluid. I bring this to attention so that it might be given further study in other areas.

DR. ELMER P. R. MAURER, Cincinnati, Ohio.—I would like to emphasize the superiority of complete extirpation technique over that of simple decortication. My

associate, Dr. Mendez, and myself during the past 5 years have preferred complete excision of the pleural pocket, regardless of the etiological background. I would like to show a few slides to illustrate the method.

(slide) This individual had a massive fibrohydrothorax of several years' duration.

(slide) This shows the complete pocket. We make the same effort as described by Dr. Samson to free the pocket in the extrapleural plane from the thoracic parietes all the way around to the midpleural reflection. We also free it from the diaphragm and then deliver the pocket with the attached lung out through the chest incision. If the lung is then placed on positive pressure it can be used as a "dissecting board" for conventional decortication.

This slide shows the pulmonary aspect of the hydrothorax pocket; (slide) the chest wall aspect; (slide) postoperative film 10 days after operation.

(slide) This is a massive infected oleothorax, instituted therapeutically in 1940 and removed by us in 1953. The tremendous size of the pocket is obvious.

(slide) A tuberculous empyema which was completely removed.

(slide) Chest wall surface of a large pocket, the etiology of which we did not know until it was completely removed.

(slide) After examination of the specimen in the laboratory, we were extremely pleased that we had used the circumferential excision technique because this lesion was due to blastomycosis.

We certainly feel that the technique for complete removal of the pleural pocket is surgically sound and attended with minimal blood loss. It obviates contamination that would be unavoidable by the conventional decortication procedure and permits more rapid and complete return of normal chest wall and lung function. Contamination in the last case presented would have been disastrous.

DR. DAVID H. WATERMAN, Knoxville, Tenn.—As the result of the work of Paul Samson and the others who re-introduced decortication during World War II, this procedure needs no defense before our Association.

I am particularly gratified that Dr. Samson has had an experience comparable to ours in false re-expansion. No residual pleural space need be present in order to do a successful decortication, as he pointed out. When we presented our work on decortication before this Association 2 years ago, we showed that progressive return of pulmonary function in such cases would continue to take place over an 18-month period.

As to empyema, I would like to again pay tribute to the late Dr. Carl Eggers, former President of this Association, for his brilliant pioneer work in decortication after World War I. He successfully performed the same operation we are doing today in both tuberculous and nontuberculous empyema without, of course, the benefit of antibiotic protection.

There was considerable interest manifested in the cases of decortication for malignancy which we included in our series 2 years ago. Of the 5 cases presented, the first was done in 1945. Since the report we have performed several cases, one of which I should like to present at this time.

(slide) The patient was a 49-year-old woman who had a 3-month history of progressive dyspnea and chest pain, and who presented herself with this pleural effusion on the right side which required repeated aspiration. Cytologic examination of the pleural fluid for malignant cells was negative. When the chest was opened to obtain a pleural biopsy, the nature of the peel was felt to warrant a decortication. Grossly, the thickened pleura was indistinguishable from that found in cases of persistent tuberculous effusion. Microscopically, it was found to be metastatic carcinoma.

(slide) This is the x-ray of the chest some 5 months after decortication. The pleural space is completely obliterated, the right lung appearing quite normal. The patient is entirely free of symptoms, has gained weight, and is working regularly. The primary site of the carcinoma is still not apparent.

(slide) This is the thick peel removed at decortication. I think you will agree that, with such a peel, the effusion would not respond to conservative therapy such as intrapleural nitrogen mustard.

ACHALASIA OF THE ESOPHAGUS: FURTHER THOUGHTS ON SURGICAL MANAGEMENT

Wm. M. Tuttle, M.D., Robert T. Crowley, M.D. (by invitation), and Raymond J. Barrett, M. D. (by invitation,) Detroit, Mich.

ACHALASIA of the esophagus is not a new disease. Purden[1] (1821) is usually thought of as the first to describe it. Hurst,[2] in 1934, stated that Willis had reported a patient, who was obviously one with achalasia, over a century and a half before Purden's report.

As was common to all disorders of that type located in portions of the body beyond the reach of surgery at that time, nothing was done. Both of the reports were made on the basis of autopsy material.

It required time to come to conclusions on an operative approach. This, apparently, came at the turn of the nineteenth and twentieth centuries.

Throughout the first decade of the twentieth century, various and sundry procedures were done, such as plication of the esophagus in an attempt to narrow the size of the lumen and, also, the removal of strips of the esophagus in the hope of the same result.[3, 4] These were not altogether successful. In 1910, Wendel[5] performed an operation in which an incision was carried through the entire layer of the lower esophagus and upper stomach at the gastrocardiac junction in a vertical line and then sutured the mucosa and the muscularis in layers horizontally. Such an operative procedure had been suggested by Marwendel[6] some years previously.

Heller[7] (1913) suggested and performed an anterior and posterior myotomy of the esophagogastric junction. The incision was carried only to the mucosa. He felt that the double incision was needed, in the fear that scarring postoperatively along the incision might well lead to a return of symptoms from further narrowing.

Heyrovsky[8] (1912) performed an esophagogastrostomy, in which the fundus of the stomach was anastomosed to the esophagus in the side-to-side line. The idea was the result of previous experimental work done on animals.

In 1915, Girard[9] performed, on 2 patients, a modification of the Wendel type of procedure, in which only the muscularis of the upper portion of the cardiogastric junction and the lower portion of the esophagus was incised

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Read at the Thirty-eighth Annual Meeting of The American Association for Thoracic Surgery at Boston, Mass., May 16-18, 1958.

down to the mucosa and then the layers were sutured in a transverse direction. This, apparently, was successful in both instances. Ferdet and Weber[10] had suggested a similar procedure some 5 years before. Apparently Girard did not report his operative approach or his results, and, consequently, the approach lay buried for a number of years.

Throughout the remaining two decades, direct approach to the disease covered in this discussion was, with the exception of sporadic attempts, given up, and the treatment for achalasia became almost routinely one of bougienage and other methods of dilating the cardiogastric sphincter area. As time advanced, it became evident that dilatation by bougienage was not always the answer to the problem and, while the results of such procedures were glowing in the earlier reports, they apparently did not completely relieve, in many instances, the individual's difficulty. In the year 1940, Ochsner and

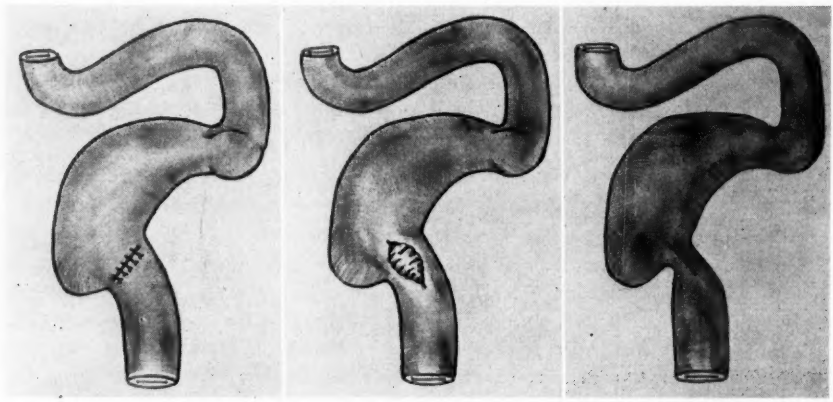

Fig. 1. Fig. 2. Fig. 3.

Fig. 1.—This drawing shows the line and position of the incision through the muscularis.

Fig. 2.—The incision through the muscularis of the esophagus and the muscularis and the serosa of the cardia of the stomach. The incision is actually somewhat longer than has been depicted by the artist.

Fig. 3.—The extramucosal layers sutured in a transverse direction.

DeBakey[11] reported 3 patients operated upon by a direct approach and gave an excellent review of the literature to that date. The paucity of cases reported from this large clinic would indicate that there was not an extensive amount of direct surgical approach to achalasia of the esophagus being done. At the conclusion of World War II, a revived interest in this problem expressed itself, and there were at that time numerous reports of various operative procedures being carried out to alleviate the obstructive process at the lower end of the esophagus. A long-time follow-up of these procedures, which, at that time, were mainly those following the pattern of Wendel and Heyrovsky, were not actually reported by the original authors, but a very definite swing to the Heller type of operative approach would lead one to believe that the results were really not as good as those brought forth in the original articles.

In our own experience during this period from 1945 until 1949, operative procedures in which the entire wall of the esophagus and the stomach were incised and then sutured in a transverse direction, or those in which a side-to-side anastomosis between the esophagus and the fundus was done, did not give satisfactory end results. The incidence of esophagitis was high and, in several instances, ulceration along the suture line led to hemorrhage. Two of these were fatal. In other patients, it was necessary to break down the anastomosis, resect the lower end of the esophagus, and do an esophagogastrostomy. Here, too, the results were not good. Regurgitation with esophagitis was frequent, and the patient was probably more uncomfortable than before the operation. It was because of this, and these unfortunate experiences, that we reverted to the procedure described by Girard in 1915. At the time the first of these were done we were not acquainted with Girard's work.

MATERIAL

Such an operative approach has now been carried out on 36 individuals. All of these people had had symptoms for a number of years, the longest being approximately 30 years. The vast majority, and in the total series during its early period of use, had been dilated on either one or numerous occasions. Later our thinking has been modified in this direction that, regardless of dilatation, if the esophagus is large and sigmoid in character, such a patient is deemed more suitable for the direct operative approach, rather than attempt at bougienage because of the dangers of splitting the esophagus or tearing it in either the introduction of the bougie or from the pressure built up in the various hydrostatic or pneumostatic bags used in this type of dilatation.

RESULTS

As was stated above, 36 patients were so operated upon. In 33, the results were good; in 2, poor; 2 are dead. No classification of "excellent" in the field of results is reported here, because it is the feeling of the authors that no result in esophageal surgery can be considered altogether excellent. The classification of "good" has been used to mean that the degree of dysphagia has been markedly or completely removed and that there is no evidence of regurgitation and esophagitis. In the 2 individuals classified as a poor result, the reason was that there was continued dysphagia in 1 and in the other there was a bothersome reflux esophagitis, which later required further operative procedures, namely, esophageal resection and esophagogastrostomy. The end result in the latter case is still a problem. The patient still has esophagitis and is, in reality, while still alive some 9 years later, a most unhappy person. The first patient classified as poor has had no further operative procedures, but has been followed with bougienage with the mercury-filled bougie. She does fairly well under this regime. A report of the 2 patients who died follows: The first died on the tenth postoperative day from a fulminating mediastinitis

as a result of perforation of the esophagus some 4 inches above the operative site. The reason for this could never be accurately determined. The second patient actually had an excellent result as far as the operative approach was concerned, but died 2½ years later from a coronary thrombosis. The remaining 32 patients are, apparently, well.

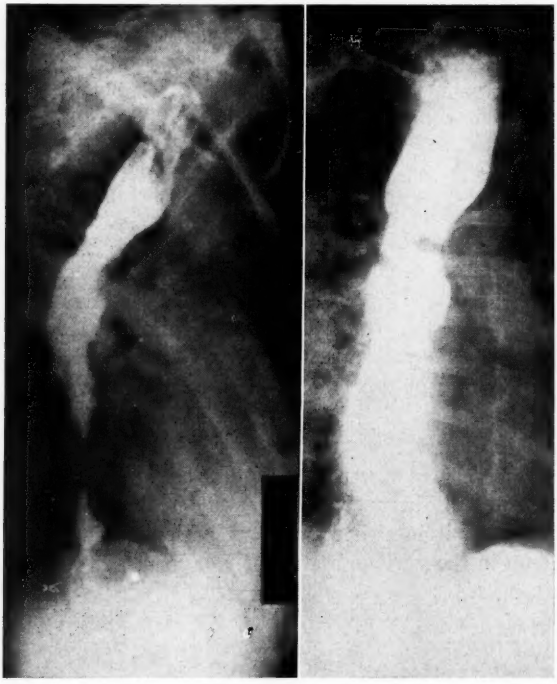

Fig. 4.—The roentgenograms reveal on the right the preoperative appearance of the esophagus. The postoperative appearance is to the left.

DISCUSSION

The true cause of achalasia of the esophagus, in the thinking of these authors, is still undetermined. Numerous etiological factors have been suggested but in no instance, according to our feeling, have they completely filled the etiological bill. A list of these causes may be seen in Table I. Within recent years, the neurological degeneration theory has seemed the most popular. Still, as regards this theory, there seems to be a rather large amount of disagreement. It has been thought that the degeneration of the plexus of Auerbach is one of the prime causes of the disease, for it has been to the action of this plexus that motility in the lower portions of the esophagus is attributed. Studies of the motility of the entire esophagus have been carried out by the use of air-filled and fluid-filled balloons. It has been demonstrated by Flood[12] and his associates that even in achalasia there is still considerable

motility in the lower end of the esophagus as recorded by their readings and by the ability of the esophagus to promote propulsion of the balloon downward. On the other hand, Olsen[13] and his associates have felt from their experimental work that the diagnosis of achalasia can be accurately made by the lack of motility in this portion of this organ. It well may be that, in

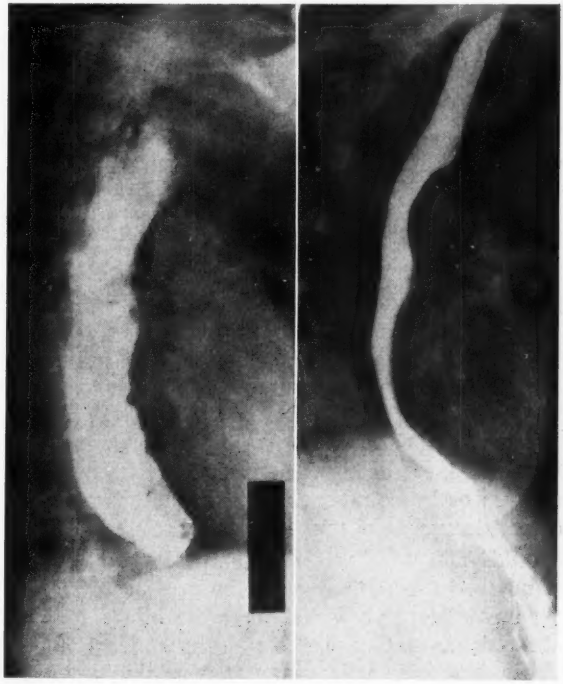

Fig. 5.—The preoperative appearance of the esophagus is revealed at the left. The postoperative view of the esophagus, made approximately 1 year later, appears at the right.

those instances in which the esophagus is markedly dilated, the size of the balloon was actually insufficient to stimulate the esophageal wall to contraction. It must be further realized that in any study of the wall of a markedly dilated esophagus, in which the number of islands of the plexus of Auerbach is counted, that the paucity of the aforementioned may be due to the greatly increased area of the esophageal wall. It would appear that there is no way

TABLE I. PROBABLE CAUSES OF ACHALASIA

1. Dysfunction of nerve supply
A. Vagal influence
B. Parasympathetic influence
C. Degeneration of plexus of Auerbach
2. Hypertrophy of the cardiac muscle
3. Hepatogastric ligament
4. Compression from other sources

to obviate this error from a statistical standpoint. The only true method is probably that in which it is adequately demonstrated that there has been actual degeneration of the plexus bodies.

In the various patients upon whom we have operated, we have been almost uniformly impressed by the fact that the musculature in the region of the cardiogastric junction is not of normal caliber; in fact, in some instances, it is markedly thickened and has the appearance of the pyloric sphincter in children with pylorospasm. This observation has led us to believe that there is much more to the theory of hypertrophy of the musculature in this area than has been previously credited. It is difficult to justify the presence of a partially atonic muscle above the sphincter and markedly hypertrophied muscle over the sphinctal area on the basis of neurological degeneration. It would seem to us that the reverse is probably true and that the muscle in this area thins out under the pressure of long retained food and mouth secretions, because of a markedly hypertrophied muscle at its lower end.

It was because of these considerations and observations, as stated in the above paragraph, that we felt that an operative approach such as has been used by us would do two things. First, it would allow the sphincter to become open so that deglutition would be possible and comfortable and free from distress; and, second, that sewing the muscle in a transverse direction would cause enfolding of the gastroesophageal mucosa in such manner as to afford a type of sphincter or stop-gap against reflux in this area. The fact that in 35 of the 36 patients there has been no reflux, as demonstrated either by the patient's symptoms or by roentgenologic examination, would lead us to feel that we are correct in our major premise.

Earlier in this article, it was mentioned that it was our feeling that, perhaps, it is unsafe to do dilatation in the presence of a markedly dilated or sigmoid-type esophagus, because the musculature of the esophagus is extremely thin and perforation is, theoretically at least, highly probable. In fact, this thought is borne out by a report from the Mayo Clinic[14] in which there were ten perforations in 550 patients dilated. This, as will be seen, is a perforation incidence of approximately 2+ per cent. With modern techniques, the fatality rate in the direct approach does not approach or exceed this level. Therefore, it has been our practice, when such an individual, namely one with a so-called megaesophagus, is encountered, even though dilatation has never been carried out, to use the direct approach rather than attempt dilatation. In the main, the patient is more satisfied with the result, and I am certain that the physician is as well. We do not mean by the former statements that we are entirely against dilatation, because that is not the case. In those individuals with a moderately dilated esophagus, but in which the esophageal wall appears to be adequate, dilatation can be carried out with a good degree of success. If it fails, then the patient is placed in the group who are thought to be benefited only by a direct approach and this has been carried out. It is an interesting fact that individuals with long-standing esophageal disease are oftentimes not exact when evaluating their

own disease. They have become so accustomed to having difficulty in swallowing that it becomes like a cross which they are willing to bear and little attention is paid to it. It is not, in many instances, until these patients are operated upon and an adequate lumen is established that they for the first time realize the true nature of the difficulties which they have been experiencing for many years. It is not uncommon after the adequate lumen has been established to have the patient, of his own volition, volunteer the information that he never realized before what it was to swallow so comfortably.

CONCLUSION

1. Thirty-six patients with achalasia of the esophagus were operated upon by us from 1949 until 1958.

2. Thirty-three of these patients have had a good result. In 2, the result was poor. Two are dead.

3. A modification of the Heller procedure has been described and has been used by the authors in the feeling that a pseudosphincter at the lower end of the esophagus is thus established and that regurgitation of gastric secretions and subsequent esophagitis is reduced.

REFERENCES

1. Purden, T.: An Extraordinary Case of Dilatation of the Esophagus, Forming a Sac, Extending From Two Inches Below the Pharynx to the Cardiac Orifice of the Stomach, London M. & Physiol. J. **46**: 540, 1821.
2. Hurst, A. F.: Some Disorders of the Esophagus, J. A. M. A. **102**: 582, 1934.
3. Meyer, W.: Cardiospasm Successfully Treated by Thoracotomy and Esophagoplication, J. A. M. A. **56**: 1437, 1911.
4. Jaffe, K.: Ueber idiopathische Oesophaguserwertereng, Müchen med. Wchnschr. **44**: 386, 1897.
5. Wendel, W.: Zur Chirurgie des Oesophagus, Arch. f. klin. Chir. **93**: 311, 1910.
6. Marwendel, G.: Die Aufklappung des Rippenbogens zur Erleichterung Operativer Eingriffe im Hypochondrium und in Zewerchfellkuppelraum, Zentralb. f. Chir. **30**: 938, 1903.
7. Heller, E.: Extramukose cardioplastik beim chronischen Cardiospasmus mit Dilatation des Esophagus, Mitt. a. d. Grenzgeb. d. Med. u. Chir. **27**: 141, 1913-1914.
8. Heyrovsky, H.: Casuistik und Therapie der idiopathischen Dilatation der Speiserohe Oesophago-gastro-anastomosis, Arch. f. klin. Chir. **100**: 703, 1912-1913.
9. Girard cited by von Hacker and Lotheissen.
10. Ferdet: Le stenose hypertrophique du pylore chez le nouveau-ne, Arch. d. mal. de l'app. digestif. **2**: 393, 1908.
11. Ochsner, A., and DeBakey, M.: Surgical Consideration of Achalasia, Arch. Surg. **41**: 1146, 1940.
12. Flood, G.: Propulsion Motility of the Esophagus in Achalasia and Other Disorders, Gastroenterology **34**: 410, 1958.
13. Olsen, A. M., Schlegel, J. F., Creamer, B., and Ellis, F. H., Jr.: Esophageal Motility in Achalasia (Cardiospasm) After Treatment, J. THORACIC SURG. **34**: 615, 1957.
14. Ellis, F.: Surgical Treatment of Cardiospasm, J. A. M. A. **166**: 29, 1958.

DISCUSSION

DR. HENRY L. HEIMLICH, New Rochelle, N. Y.—We have been successfully using a reversed gastric tube for replacing the esophagus. In this procedure, which is being shown in detail in the motion picture section of this meeting, a tube is created from the greater curvature of the stomach. The tube, which remains attached near the cardia, is

reversed in direction, and brought to the neck subcutaneously, retrosternally, or intrapleurally. The gastric tube is anastomosed to the cervical esophagus. Peptic esophagitis does not occur, because the antrum of the stomach, which does not secrete acid, forms the part of the tube that is anastomosed to the esophagus. This has been confirmed clinically in patients followed up to 7 years by Dr. Dan Gavriliu. Microscopic sections through the esophagogastric junction showed no evidence of inflammation in the esophagus in those of my own patients who have died of metastatic carcinoma several months after operation. I would like to suggest that patients with peptic esophagitis or achalasia, when other procedures have failed, and patients with bleeding esophageal varices, may be benefited from esophageal replacement with a reversed gastric tube. In these cases, a shorter reversed gastric tube than that used in the treatment of carcinoma may be the procedure of choice.

DR. MARK M. RAVITCH, Baltimore, Md.—Dr. Tuttle's interesting paper touched on the question of the hiatus which existed for some 30 odd years after Heller's work, until we all again became aware of it through the paper of Barrett and Franklin, some 10 or 12 years ago. Dr. Felix Steichen and I were interested in trying to find the reason for this temporary eclipse of what seems to be a good operation. As Dr. Tuttle said, Gottstein from Mikulicz' Clinic in 1901 suggested the operation, but did not perform it, and it was Girard who actually did perform the first successful extramucosal cardiomyotomies. This Girard of Berne was not, Mr. President, with reference to your remarks of this afternoon, a specialized surgeon. Among other things, he performed two successful hemipelvectomies before anyone else had done so.

We then tried to write to Heller's scientific descendents and were surprised after some time (slide) to get a letter from the Russian Zone from Dr. Heller, who sent this photograph of himself that he was writing from Leipzig, "wo ich doch labe'' using this form rather than "wohne." Dr. Heller said that he had never had any misgivings about the operation, that he had never ceased to perform it and with continually good results, but that he had never been able to convince his own countrymen of its value, whereas the French had taken up the operation at once and had continued to perform it ever since. As Dr. Tuttle and many of you know, Santy of Lyons, D'Allaines of Paris, and his pupil, Lortat-Jacob, have enormous personal series of the Heller operation. They raised the point, and in conversation with Mr. Phillip Allison this point was raised again, that poor results after the Heller procedure, particularly esophagitis, are probably due to the presence of a hiatus hernia, either pre-existing and unsuspected, or one produced in the course of operative manipulation. The French go so far as to suggest that the hiatus should regularly be tightened and that, perhaps, frequently the stomach should be sutured back to the esophagus, reconstructing the acute angle of His.

DR. RICHARD H. SWEET, Boston, Mass.—There is not very much time left at this session so I will confine my remarks to just a few comments. I have used the Heller operation with preeminent success. I have known of Dr. Tuttle's interest in this procedure, the Girard operation. I have not felt, in my experience at least, that I have needed it, because I have not been aware of the results of regurgitation and esophagitis and so on. On the other hand, when we stop to think about it, there are two distinct anatomic types of achalasia of the esophagus; the type of which Dr. Tuttle showed two examples is the type on which I always use the Heller operation. In that type, the lower end of the esophagus is characterized by preeminently thickened, almost sphincter-like, hypertrophied circular muscle. I think, though, that I tend to make an incision through the muscularis even longer than Dr. Tuttle does, and I would be a little fearful about restoring the anatomic relation.

On the other hand, in the other type of achalasia, which is characterized usually by the enormous dilatation of the esophagus with its sigmoid tortuosities, we usually find, as you know, a very thin-walled atrophic distal end proximal to the cardia. (Of course this disease has nothing to do with the cardia whatsoever). In that group, it

seems to me that this suggestion of Dr. Tuttle's might be very valuable indeed, because it would overcome any temptation to open the lumen and suture the other way. This, it seems to me, might be ideal.

As Dr. Ravitch said, I, too, have found hiatus hernia in a number of these patients, and have made a point of repairing it, and that may be a factor in our not seeing regurgitation following our use of the Heller operation.

DR. LAMAR SOUTTER, Boston, Mass.—I think this is a very interesting paper of Dr. Tuttle's. I would like to ask him one question, that is, whether he has had any difficulty in suturing that thickened muscle transversely. It is also well worth while to re-emphasize one point, that is, that the Heller operation consists of both an anterior and a posterior incision in the muscularis. Most people in this country think it is only an anterior incision. Recently, in fact, someone suggested to me that he was going to modify the Heller operation by doing it anteriorly and posteriorly as well. I have done a number of modified Heller operations with reasonably good results. There is, however, a simpler effective method of curing achalasia which I think should not be overlooked.

(slide) That is the use of some form of internal rupture of the circumferential muscle fibers by means of an expanding pneumatic bag. The difference between this type of dilatation and that with a bougie is that the bougie is smaller in circumference (a No. 45 is the largest which can readily be passed and measures 45 mm. in circumference over but a short area), whereas the bag measures 107 mm. for a length of 15 mm. when fully expanded.

(slide) The bag should not be used on anyone who has scarring inflammation, or carcinoma of the esophagus or the results may be disastrous. When we place the bag in position under the fluoroscope, we take a spot film which you can see here, showing the bag indented by the hypertrophied muscularis.

After expanding the bag three times at intervals of a minute to a pressure of 15 pounds, we retake the spot film to show (slide) that it is in the correct position and no longer indented. What happens to the crura with this treatment? I do not know. A gastrointestinal series taken before discharge from the hospital is necessary to demonstrate the ready passage of barium into the stomach.

(slide) I thought this might impress you to show the difference in the length of time these patients are in the hospital and the hospital cost depending on whether they are treated with the bag or by the Heller operation. For the simpler procedure, the stay is one third as long and the cost less than one quarter of what it is for the Heller operation. The Blue Shield charge is $10 for the former, $150 for the latter. We have, on a brief follow-up, had good results in 10 out of 12 patients.

MR. R. H. R. BELSEY, Bristol, England.—Dr. Blades, I should like to thank you very much indeed for the privilege of being a guest at this meeting. This has been a very interesting discussion and I think the consensus is that the Heller operation is an excellent one. Dr. Ravitch and Dr. Sweet stressed the importance of the co-existence of an hiatus hernia with achalasia; in these cases, relief of the obstruction by a Heller procedure will most probably lead to esophagitis. Dr. Sweet has also stressed the point that the majority of people now make a longer vertical incision through the esophageal muscle, and to perform the operation from above involves some mobilization of the cardia. If this mobilization is carried out with gay abandon it may lead to the development of an hiatus hernia later, unless the normal valvular mechanism at the cardia is restored after the Heller procedure. Failure in the past to undertake this step has, to my mind, been one of the main reasons for the development of postoperative esophagitis, and failure of the operation.

I was sorry that nobody raised the important problem of carcinoma complicating achalasia. In my own series of 54 cases of achalasia, there have been 7 cases of squamous-cell carcinoma, an incidence of about 12 per cent. Three of the patients were found to have growths while being investigated for the achalasia; it is an interesting fact that

the other 4 patients had had Heller operations performed 18 months to 4½ years previously. The growth may well have been present at the time of the original operation, but obscured by the retention of esophagitis.

What moral is to be drawn from all this I do not know, unless it be that one should try to get cases earlier by emphasizing to physicians the premalignant nature of the retention esophagitis that complicates achalasia, and the importance of early control of the esophagitis by prompt relief of the obstruction.

DR. TUTTLE (Closing).—In answer to one question asked—in the 2 cases shown, both patients had been dilated on numerous occasions over considerable periods; 1, for 15 years and 1 for 17 years, probably on an average of about six or eight times a year. Now, six or eight times ten times 15, exceeds the $450 fee which was the surgeon's charge, so actually they are not getting off too well with the $10 dilatation fee. I have one of those bags, too, and I use it on occasions. I do not think that any patient with a single achalasia should be operated upon until dilatation has been given a chance. In the so-called megalo-esophagus I do not believe it is safe to try to dilate that type of lesion, because of the possibility of rupture, and even in a clinic where they have expert endoscopists such as the Mayo Clinic, they have 2 per cent perforations average in one report.

I want to thank Dr. Belsey for discussing the paper, and also the other discussers.